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20, HANOVER SQUARE, W.;
October 10th, 1902.

228204

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1882 BARKER, FREDERICK CHARLES, M.D., Surgeon-Major, Bombay Medical Service, India.

1894 BARLING, HARRY GILBERT, 85, Edmund Street, Birmingham. (C. 1901– .) *Trans.* 3.

1875 BARLOW, SIR THOMAS, Bart., K.C.V.O., M.D., 10, Wimpole Street, W. (C. 1880–2, 1891–4, S. 1888–90, V.P. 1895–8.) *Trans.* 11, *C.C.* 5.

1902 BARNARD, H. L., 25, Welbeck Street, Cavendish Square, W. *Trans.* 1, *C.C.* 5.

1888 BARRS, A. G., M.D., 22, Park Place, Leeds.

1888 BARTON, EDWIN A., 35, Cheniston Gardens, Kensington, W.

1891 BARTON, J. KINGSTON, 2, Courtfield Road, S.W. (C. 1901– .)

1901 BARWELL, HAROLD SHUTTLEWORTH, 55, Wimpole Street, Cavendish Square, W.

O.M. BARWELL, RICHARD, 55, Wimpole Street, W. (C. 1872–5, V.P. 1877–9.) *Trans.* 21, *C.C.* 2.

O.M. **BASTIAN**, HENRY CHARLTON, M.D., F.R.S., 8A, Manchester Square, W. (C. 1876–8, V.P. 1891–3.) *Trans.* 7.

1896 BATTEN, FREDERICK E., M.D., 33, Harley Street, W. *Trans.* 3, *C.C.* 3.

1902 BATTEN, GEORGE BECKETT, M.D., Glenmore, 2, Underhill Road, Lordship Lane, S.E.

1899 BATTEN, RAYNER DERRY, M.D., B.S., 33, Harley Street, W. *C.C.* 1.

1888 BATTERHAM, JOHN WILLIAMS, M.B., B.S., Bank House, Grand Parade, St. Leonard's.

1886 BATTLE, WILLIAM HENRY, 49, Harley Street, W. (C. 1899–1902.) *Trans.* 7, *C.C.* 12.

1868 BÄUMLER, CHRISTIAN G. H., M.D., University of Erlangen. *Trans.* 4.

1896 BEATON, ROBERT MILNE, M.B., 7, Dartmouth Park Road, N.W.

1898 BEDDARD, ARTHUR PHILIP, M.B., 44, Seymour Street, Portman Square, W.

1880 **BEEVOR**, CHARLES EDWARD, M.D., 135, Harley Street, W. (C. 1890–1.) *Trans.* 2, *C.C.* 9.

1901 BEEVOR, SIR HUGH REEVE, Bart., M.D., 17, Wimpole Street, Cavendish Square, W. *C.C.* 2.

1884 BENHAM, F. LUCAS, M.D., Campden House, nr. Exeter, Semaphore, South Australia. *Trans.* 4.

ELECTED

1883 BENHAM, ROBERT FITZROY, 11, Earl's Court Square, S.W.

1874 BENNETT, Sir WILLIAM HENRY, K.C.V.O., 1, Chesterfield Street, W. (C. 1889, 1892–4, S. 1890–1, V.P. 1895–8.) *Trans.* 4, *C.C.* 3.

1896 BENTHAM, EDWARD COOPER, 10, Princes Street, Cavendish Square, W.

1889 BENTLEY, ARTHUR J. M., M.D., Mena House, Pyramids, Cairo, Egypt.

1896 BERKELEY, G. H. A. COMYNS, M.B., 53, Wimpole Street, W.

1882 BERRY, FREDERICK HAYCRAFT, M.B., Watford, Herts.

1885 BERRY, JAMES, B.S., 21, Wimpole Street, W. (C. 1898–1901.)

1889 BIDWELL, LEONARD ARTHUR, 15, Upper Wimpole Street, W. (C. 1901– .) *Trans.* 5, *C.C.* 1.

1882 BINDLEY, PHILIP HENRY, M.B. (Travelling).

1889 BISSHOPP, FRANCIS ROBERT BRYANT, M.A., M.B., B.S., Belle Vue, Mount Pleasant, Tunbridge Wells.

1893 BOND, JAMES WILLIAM, M.D., 26, Harley Street, W. *C.C.* 2.

1898 BOSANQUET, WILLIAM CECIL, M.D, 117A, Harley Street, Cavendish Square, W.

1888 BOSTOCK, ROBERT ASHTON, Cefn Mor, Penmaen, Glamorganshire.

1900 BOUSFIELD, ARTHUR, M.B., The Cheal, Hornsey Lane, N.

1899 BOWLAN, MARCUS MARWOOD, M.B., B.S., The Infirmary, Raine Street, St. George-in-the-East, E.

1883 BOWLBY, ANTHONY A., C.M.G. (*Hon. Secretary*), 24, Manchester Square, W. (C. 1891–3.) *Trans.* 3, *C.C.* 4.

1883 Bowles, ROBERT LEAMON, M.D., 16, Upper Brook Street, W. (C. 1890–1.)

1896 BOX, CHARLES R., M.D., 2, Devonshire Place, Portland Place, W. *Trans.* 1.

1889 BOYD, STANLEY, B.S., 134, Harley Street, W. (C. 1899–1902.) *Trans.* 1, *C.C.* 7.

1890 BRADFORD, JOHN ROSE, M.D., F.R.S., 8, Manchester Square, W. (C. 1900–1902.) *Trans.* 2.

1878 BRIDGES, ROBERT, M.B., M.A., The Manor House, Yattendon, Berkshire. *Trans.* 1.

1868 BRIGHT, GEORGE CHARLES, M.D., Cannes, Alpes Maritimes, France.

O.M. BROADBENT, Sir WILLIAM HENRY, Bart., K.C.V.O., M.D., F.R.S., 84, Brook Street, W. (C. 1871–3, V.P. 1881–3, P. 1887–8.) *Trans.* 17.

1887 BROCK, J. H. E., M.D., B.S., 77, Fellows Road, South Hampstead, N.W.

1894 BRODIE, C. GORDON, Fernhill, Wootton Bridge, Isle of Wight. *C.C.* 1.

1898 Brook, WILLIAM HENRY BREFFIT, M.D., B.S., 8, Eastgate, The Close, Lincoln. *Trans.* 1.

1890 BROWN, WALTER HENRY, 19, Queen Street, Leeds. *Trans.* 2.

1876 BROWNE, GEORGE BUCKSTON, 80, Wimpole Street, W. (C. 1891–3.) *Trans.* 3.

1893 BROWNE, JAMES WILLIAM, M.B., 37, Holland Park Avenue, W. *Trans.* 1.

ELECTED

1887 BROWNE, OSWALD AUCHINLECK, M.A., M.D., 7, Upper Wimpole Street, W.

1883 BRUCE, JOHN MITCHELL, M.D., 23, Harley Street, W. (C. 1901– .)

1893 BRYANT, JOHN HENRY, M.D., 4, St. Thomas's Street, S.E., and 8, Mansfield Street, Portland Place, W. *Trans.* 2.

O.M. BRYANT, THOMAS, M.Ch., 27, Grosvenor Street, W. (C. 1872, V.P. 1876–7, P. 1885–6.) *Trans.* 9.

1890 BUCKLAND, FRANCIS O., B.A., M.B., C.M., Brockenhurst, Hants. *Trans.* 1.

1901 BUCKNALL, THOMAS RUPERT HAMPDEN, M.S., 35, Harley Street, W.

1891 BURGHARD, FREDERIC FRANÇOIS, M.D., M.S., 86, Harley Street, W.

1881 BURNET, ROBERT WILLIAM, M.D., 36, Grosvenor Street, W. *Trans.* 1.

1879 BURTON, WILLIAM EDWARD, 24, Wimpole Street, W.

1900 BUSCH, JOSEF PAUL ZUM, M.D., Chiswell House, 133, Finsbury Pavement, E.C.

1881 BUTLIN, HENRY TRENTHAM, 82, Harley Street, W. (C. 1887–9.) *Trans.* 7, *C.C.* 1.

1884 BUXTON, DUDLEY WILMOT, M.D., B.S., 82, Mortimer Street, W.

1902 BUZZARD, EDWARD FARQUHAR, M.B., 33, Harley Street, W.

O.M. BUZZARD, THOMAS, M.D., 74, Grosvenor Street, W. (S. 1870–2, C. 1873–6, V.P. 1880–1, P. 1895–7.) *Trans.* 15; *C.C.* 3.

1901 CADDY, ADRIAN, M.B, St. George's Hospital, Hyde Park Corner, S.W.

1891 CADDY, ARNOLD, 2/2, Harington Street, Calcutta.

1893 CALEY, HENRY ALBERT, M.D., 24, Upper Berkeley Street, W. *C.C.* 1.

1890 CALVERT, JAMES, M.D., The Warden's House, St. Bartholomew's Hospital, E.C. *Trans.* 2, *C.C.* 2.

1893 CAMPBELL, HARRY, M.D., 23, Wimpole Street, W., and Coniston Cottage, Fox Lane, Upper Norwood, S.E. *C.C.* 3.

1891 CARLESS, ALBERT, M.S., M.B., 10, Welbeck Street, W.

1890 CARR, JOHN WALTER, M.D., 19, Cavendish Place, W. *Trans.* 1, *C.C.* 6.

1901 CARSON, HERBERT WILLIAM, 26, Welbeck Street, Cavendish Square, W.

1901 Carwardine, THOMAS, M.S., 16, Victoria Square, Clifton, Bristol. *Trans.* 1.

1885 CATON, RICHARD, M.D., 86, Rodney Street, Liverpool. *Trans.* 3.

O.M. CAYLEY, WILLIAM, M.D., 27, Wimpole Street, W. (C. 1874–5, S. 1876–8, C. 1879–80, V.P. 1885–6.) *Trans.* 8, *C.C.* 3.

1890 CHAPMAN, CHARLES WILLIAM, M.D., 21, Weymouth Street, W. (C. 1900– .) *Trans.* 2, *C.C.* 2.

1884 CHAPMAN, PAUL M., M.D., 1, St. John's Street, Hereford. *C.C.* 1.

1893 CHARSLEY, ROBERT STEPHEN, "The Old Barn," Slough.

1885 CHEYNE, W. WATSON, M.B., C.M., F.R.S., 75, Harley Street, W. (C. 1894–6.) *Trans.* 2, *C.C.* 8.

1873 CHISHOLM, EDWIN, M.D., 44, Roslyn Gardens, Darlinghurst, Sydney, New South Wales.

ELECTED

O.M. **CHURCH**, Sir WILLIAM SELBY, Bart., K.C.B., M.D., 130, Harley Street, W. (C. 1874–6.)

1873 CHURTON, THOMAS, M.D., 35, Park Square, Leeds. (C. 1889–91.) *Trans.* 6.

O.M. CLAPTON, EDWARD, M.D., Towercroft, 41, Eltham Road, Lee, S.E. (C. 1872–4.) *Trans.* 1.

1874 CLARK, ANDREW, 71, Harley Street, W. *Trans.* 1.

1887 CLARK, FRANCIS WILLIAM, Hong-Kong.

1898 CLARKE, JAMES JACKSON, M.B., 18, Portland Place, W. *Trans.* 1, *C.C.* 6.

1885 Clarke, J. MICHELL, M.B., 28, Pembroke Road, Clifton, Bristol.

1885 CLARKE, WILLIAM BRUCE, M.B., 51, Harley Street, W. (C. 1894–7.) *Trans.* 2, *C.C.* 2.

1877 Clay, ROBERT HOGARTH, M.D., 4, Windsor Villas, Plymouth.

1887 CLEMOW, ARTHUR HENRY WEISS, M.D., C.M., 101, Earl's Court Road, W. *Trans.* 1, *C.C.* 1.

1877 **CLUTTON**, HENRY HUGH, M.A., 2, Portland Place, W. (C. 1885–7, V.-P. 1902– .) *Trans.* 12, *C.C.* 3.

1898 COBB, ERNEST HERBERT, Belmont, Stevenage, Herts.

1893 COLE, ROBERT HENRY, M.D., Moorcroft, Hillingdon, Middlesex.

1882 COLLIER, HERBERT, M.D., The Grange, Gorleston, Great Yarmouth, Norfolk.

1896 COLMAN, WALTER STACY, M.D., 9, Wimpole Street, W. *Trans.* 2, *C.C.* 3.

1882 COLQUHOUN, DANIEL, M.D., Dunedin, New Zealand.

1868 COOPER, FRANK W. (address uncommunicated).

1899 COOPER, HARRY, M.D., Fownhope, Surbiton Hill, Surrey.

1902 CORNER, EDRED MOSS, B.Sc., M.B., St. Thomas's Hospital, Albert Embankment, S.E.

1880 COTTLE, WYNDHAM, M.D., 39, Hertford Street, W.

O.M. COUPER, JOHN, 80, Grosvenor Street, W. (C. 1874.)

1875 COUPLAND, SIDNEY, M.D., 16, Queen Anne Street, W. (S. 1882–4, C. 1885–7, V.-P. 1898–1901.) *Trans.* 5, *C.C.* 3.

1886 COUSINS, JOHN WARD, M.D., Riversdale, Kent Road, Southsea. *Trans.* 1.

1882 COXWELL, C. F., M.B., Brighton, Melbourne, Victoria. *Trans.* 2.

1897 CRAWFURD, RAYMOND H. P., M.D., 71, Harley Street, Cavendish Square, W. *C.C.* 2.

1879 CRIPPS, WILLIAM HARRISON, 2, Stratford Place, W. (C. 1886–8.) *Trans.* 3.

1872 CRITCHETT, Sir G. ANDERSON, 21, Harley Street, W.

1877 CROCKER, HENRY RADCLIFFE, M.D., 121, Harley Street, W. (C. 1884–5.) *Trans.* 16.

1896 CROOKE-LAWLESS, W. R., Surg.-Maj. A.M.S., M.D., Guards' Club, S.W.

1896 CROSSE, WILLIAM HENRY, M.D., 27, Albemarle Street, Piccadilly, W.

1898 CROUCH, HERBERT CHALLICE, 31, Welbeck Street, Cavendish Square, W.

ELECTED

1890 CROWLE, THOMAS HENRY RICKARD, 35, St. James's Place, S.W.
1893 CURTIS, HENRY JONES, M.D., B.S., 15, Harley Street, W. *C.C.* 1.
1872 DALBY, Sir WILLIAM BARTLETT, M.B., 18, Savile Row, W. (C. 1879–81.) *Trans.* 4.
1896 DALTON, NORMAN, M.D., 4, Mansfield Street, Portland Place, W. *Trans.* 2, *C.C.* 7.
1898 DARLEY-HARTLEY, WILLIAM, M.D., Seapoints, Cape Town, South Africa.
1893 DAUBER, JOHN HENRY, M.B., B.Ch., 29, Charles Street, Berkeley Square, W.
1886 DAVIES, ARTHUR T., M.D., 23, Finsbury Square, E.C. (C. 1897–1900.) *C.C.* 13.
1879 Davy, HENRY, M.D., 29, Southernhay, Exeter.
1889 DEAN, HENRY PERCY, M.B., 69, Harley Street, W.
1897 DEANE, JOHN HENRY, 50, Wallwood Road, Leytonstone, Essex.
1899 DE CHAZAL, EDMOND LUCIEN, M.D., Port Louis, Mauritius.
1879 Dennis, FREDERIC S., M.D., 542, Madison Avenue, New York, U.S.A.
1875 DENT, CLINTON T., 61, Brook Street, W. (C. 1884–6.) *Trans.* 5. *C.C.* 1.
O.M. DICKINSON, WILLIAM HOWSHIP, M.D., 9, Chesterfield Street, W. (C. 1874–5, V.P. 1888–9.) *Trans.* 2.
1891 DICKINSON, WILLIAM LEE, M.D., 9, Chesterfield Street, W. (C. 1902– .) *Trans.* 8, *C.C.* 1.
1894 Dickson, THOMAS HUGH, M.B., B.C., H.M. Customs, Lower Thames Street, E.C.
1871 DIVER, EBENEZER, M.D., "Garstone," St. Margaret's-on-Thames, Twickenham. (C. 1890–2.) *Trans.* 1.
1901 DOUGLAS, JOHN J., M.D., 42, Central Hill, Norwood, S.E.
1868 DRAGE, CHARLES, M.D., Hatfield, Herts.
1896 DREW, DOUGLAS, M.D., B.S., 49, Devonshire Street, Portland Place, W.
O.M. DUCKWORTH, Sir DYCE, M.D., LL.D. 11, Grafton Street, Bond Street, W. (C. 1875–7, V.P. 1887–8, P. 1891–2.) *Trans.* 20, *C.C.* 7.
1884 DUKE, EDGAR, 11, Wilbury Road, Hove.
1869 DUKE, OLLIVER THOMAS, M.B., Surgeon, Bengal Army, India.
1899 DUNCAN, ANDREW, M.D., 24, Chester Street, Grosvenor Place, S.W.
1889 DUNCAN, JOHN, M.D., St. Petersburg.
1889 DUNN, LOUIS ALBERT, M.S., 51, Devonshire Street, Portland Place, W. (C. 1899–1902.) *Trans.* 2, *C.C.* 2.
1884 EDMUNDS, WALTER, M. C., 2, Devonshire Place, Portland Place, W. (C. 1902– .) *Trans.* 2.
1899 EÏD, ALFRED, M.D., Cairo, Egypt.
1902 ELLIOT, ANDREW, M.D., 11, Upper Berkeley Street, Portman Square, W.
1882 EMOND, EMILE, M.D., 113, Boulevard Beaumarchais, Paris.
1868 EVANS, JULIAN, M.B., 123, Finborough Road, Redcliffe Square, S.W.

ELECTED

1893 EVANS, WILLMOTT H., M.D., B.S., 2, Upper Wimpole Street, Cavendish Square, W. *C.C.* 1.

1888 **EVE,** FREDERIC S., 125, Harley Street, Cavendish Square, W. (C. 1899–1902.) *Trans.* 4, *C.C.* 10.

1877 EWART, WILLIAM, M.D., 33, Curzon Street, W. (C. 1884–6.) *Trans.* 4, *C.C.* 4.

1899 FAGGE, CHARLES HERBERT, M.S., 22, St. Thomas's Street, London Bridge, S.E.

1868 **Fairbank,** FREDERICK ROYSTON, M.D., Westcott, Dorking, Surrey. *Trans.* 2.

1889 FARDON, EDWARD ASHBY, Middlesex Hospital, W.

1900 FAWCETT, JOHN, M.D., 26, St. Thomas's Street, London Bridge, S.E. *Trans.* 1.

1885 FENN, EDWARD LIVEING, M.D., Grey Friars, Colchester.

1902 FENNELL, CHARLES HENRY, M.B., 10, Devonshire Street, Portland Place, W. *C.C.* 1.

1887 FENWICK, E. HURRY, 14, Savile Row, W. *Trans.* 2, *C.C.* 2.

1872 FENWICK, J. C. J., M.D., Long Framlington, Morpeth.

1893 FENWICK, WILLIAM SOLTAU, M.D., 8, Devonshire Street, W. *Trans.* 2.

1878 FIELD, GEORGE P., 34, Wimpole Street, W.

1876 FINLAY, DAVID WHITE, M.D., 2, Queen's Terrace, Aberdeen. (C. 1885–7, S. 1891.) *Trans.* 7.

1894 FLETCHER, HERBERT MORLEY, M.D., 98, Harley Street, W. *Trans.* 1.

1878 **FONMARTIN,** HENRY DE, M.D., 26, Newberry Terrace, Lower Bullar Street, Nichols Town, Southampton.

1889 FORBES, DANIEL MACKAY, 32, Oakfield Road, West Croydon.

1890 FOSTER, MICHAEL G., M.B., M.A., Villa Camilla, San Remo, Italy.

1898 FOWLER, CHARLES OWEN, M.D., Cotford House, Thornton Heath, Surrey.

1881 FOWLER, JAMES KINGSTON, M.D., 35, Clarges Street, W. (C. 1887–9, 1899–1902, S. 1896–9.) *Trans.* 4, *C.C.* 6.

1886 FOX, R. HINGSTON, M.D., 23, Finsbury Square, E.C. *C.C.* 1.

1878 FOX, THOMAS COLCOTT, M.B., 14, Harley Street, W. (C. 1888–90.) *Trans.* 7, *C.C.* 2.

1893 FOXWELL, ARTHUR, M.D., 22, Newhall Street, Birmingham.

1902 FRENCH, HERBERT, M.B., 26, St. Thomas's Street, London Bridge, S.E.

1897 FRIPP, ALFRED DOWNING, C.B., M.V.O., M.S., 19, Portland Place, W. *Trans.* 3.

1901 FRITH, WALTER S., M.B., The Limes, Rickmansworth, Herts.

1899 FÜRTH, KARL, M.D., 94, Harley Street, W.

1890 FULLER, HENRY ROXBURGH, M.D., 45, Curzon Street, W.

1897 FURNIVALL, PERCY, 28, Weymouth Street, Portland Place, W.

1891 FYFFE, W. KINGTON, M.B., B.C., 19, Duke Street, Manchester Square, W. (Travelling, New Zealand.) *Trans.* 1.

1888 GAGE-BROWN, CHARLES HERBERT, M.D., 85, Cadogan Place, S.W.

ELECTED

1895 GALLOWAY, JAMES, M.D., 54, Harley Street, W.

1887 GARROD, ARCHIBALD EDWARD, M.A., M.D., 9, Chandos Street, Cavendish Square, W. (C. 1898–1901.) *Trans.* 4, *C.C.* 3.

1879 GARSTANG, THOMAS WALTER HARROPP, Englefield, Delamer Road, Bowdon, Cheshire.

1885 GIBBONS, ROBERT ALEXANDER, M.D., 29, Cadogan Place, S.W. *Trans.* 1.

1893 GIBBS, CHARLES, 115, Harley Street, W.

1875 GILBART-SMITH, T., M.A., M.D., 68, Harley Street, W. (C. 1883–5.)

1868 GLOVER, JAMES GREY, M.D., 25, Highbury Place, N. (C. 1878–80. V.P. 1892–4.) *Trans.* 2.

1893 GLOVER, LEWIS G., M.B., B.C., 17, Belsize Park, Hampstead, N.W.

1875 GODLEE, RICKMAN JOHN, M.S., 19, Wimpole Street, W. (C. 1882–3, 1887–9, S. 1884–6, V.P. 1895–8.) *Trans.* 14, *C.C.* 10.

1901 GOFFE, ERNEST GEORGE LEOPOLD, Western Hospital, Seagrave Road, Fulham, S.W.

1882 GOLDIE, ROBERT WILLIAM, Hirst House, Hirst, Morpeth, Northumberland.

1878 GOLDING-BIRD, C. H., M.B., 12, Queen Anne Street, W. (C. 1887–9.) *Trans.* 18, *C.C.* 1.

1894 GOODALL, EDWARD WILBERFORCE, M.D., Eastern Hospital, Homerton, N.E. *Trans.* 5.

1875 GOODHART, JAMES FREDERIC, M.D., 25, Portland Place, W. (C. 1880–2, V.P. 1897–1900.) *Trans.* 14, *C.C.* 2.

1891 GOODMAN, ROGER NEVILLE, M.B., Elmside, Kingston-on-Thames.

1869 GOODRIDGE, HENRY FREDERICK AUGUSTUS, M.D., 10, Brock Street, Bath.

1882 GOODSALL, D. H., 17, Devonshire Place, W.

1877 GOULD, A. PEARCE, M.S., 10, Queen Anne Street, W. (C. 1885–7, 1895–8, S. 1892–5, V.-P. 1898–1901.) *Trans.* 14, *C.C.* 9.

1901 GOWAN, BOWIE CAMPBELL, Great Stanmore, Middlesex.

1875 GOWERS, Sir WILLIAM RICHARD, M.D., F.R.S., 50, Queen Anne Street, W. (C. 1881–2.) *Trans.* 5.

1891 GRANT, J. DUNDAS, M.D., 18, Cavendish Square, W.

1868 GREEN, T. HENRY, M.D., 74, Wimpole Street, W. (C. 1877–9.) *Trans.* 2, *C.C.* 1.

1875 Greenfield, WILLIAM SMITH, M.D., 7, Heriot Row, Edinburgh. (C. 1881.) *Trans.* 3.

1893 **GRIFFITH**, WALTER SPENCER ANDERSON, M.D., 96, Harley Street, W.

1895 Grube, KARL, M.D., Neuenahr, Germany. *Trans.* 1.

1895 GUTHRIE, LEONARD GEORGE, M.D., 15, Upper Berkeley Street, W. *C.C.* 7.

1887 HABERSHON, SAMUEL HERBERT, M.D., 88, Harley Street, W. (C. 1901– .) *C.C.* 1.

1900 HADLEY, WILFRED J., M.D., 58, Harley Street, Cavendish Square, W.

1875 HALE, C. D. B., 3, Sussex Place, W. *Trans.* 1.

ELECTED

1878 HALL, F. DE HAVILLAND, M.D., 47, Wimpole Street, W. (C. 1885–7.) *Trans.* 8, *C.C.* 5.

1889 HALSTEAD, GEORGE EZRA, M.D., B.S., Albion Hill House, Ramsgate.

1888 HANDFIELD-JONES, MONTAGU, M.D., 35, Cavendish Square, W.

1886 **Handford**, HENRY, M.D., 6, Regent Street, Nottingham. (C. 1893–4.) *Trans.* 8, *C.C.* 1.

1886 HARDIE, JAMES, M.D., 15, St. John Street, Manchester.

1890 HARPER, JAMES, M.D., 25, Rosary Gardens, South Kensington, S.W.

1872 HARRIS, HENRY, M.D., Trengweath, Redruth, Cornwall.

1889 HARRIS, HERBERT ELWIN, M.B., 13, Lansdown Place, Victoria Square, Clifton, Bristol. *Trans.* 1.

1881 HARRISON, CHARLES EDWARD, M.B., Grenadier Guards Hospital, Rochester Row, S.W.

1892 HARRISON, DAMER, 53, Rodney Street, Liverpool. *Trans.* 1.

1869 **HAWARD**, J. WARRINGTON, 57, Green Street, Grosvenor Square, W. (C. 1876–8, 1884–6, S. 1881–3, V.P. 1888.) *Trans.* 15.

1899 **Hawkes**, CLAUDE SOMERVILLE, Gleneairn, Wickham Terrace, Brisbane, Queensland, Australia.

1886 HAWKINS, FRANCIS HENRY, M.D., 73, London Street, Reading. (C. 1902– .) *Trans.* 4, *C.C.* 3.

1889 HAWKINS, HERBERT PENNELL, M.D., B.Ch., 56, Portland Place, W. (C. 1900– .) *Trans.* 1.

1890 HAWKINS-AMBLER, GEORGE ARTHUR, 30, Rodney Street, Liverpool.

1900 HAYWARD, JOHN ARTHUR, M.D., 17, Lingfield Road, Wimbledon, S.W.

1879 HENDERSON, GEORGE COURTENAY, M.D., Kingston, Jamaica, West Indies.

1884 HERRINGHAM, WILMOT PARKER, M.D., 40, Wimpole Street, W. (C. 1894–7.) *Trans.* 6, *C.C.* 3.

1888 **Hetherington**, GEORGE HAYNES, 3, Museum Street, Ipswich.

1897 HILLIER, ALFRED P., M.D., C.M., 30, Wimpole Street, W.

1874 HOLDERNESS, WILLIAM BROWN, 15, Park Street, Windsor.

1868 **Holman**, CONSTANTINE, M.D., 26, Gloucester Place, Portman Square, W. (C. 1894–7.)

O.M. HOLMES, TIMOTHY, 6, Sussex Place, Hyde Park, W. (C. 1867–9, V.P. 1873–5.) *Trans.* 16.

1878 HOOD, DONALD WILLIAM CHARLES, M.D., 43, Green Street, Park Lane, W. (C. 1891–3.) *Trans.* 1.

1884 HORSLEY, Sir VICTOR, M.B., F.R.S., 25, Cavendish Square, W. (C. 1894.) *Trans.* 2, *C.C.* 1.

1901 HORTON-SMITH, PERCIVAL, M.D., 15, Upper Brook Street, Grosvenor Square, W.

1895 HOUGH, CHARLES HENRY, Full Street, Derby.

1880 **HOVELL**, T. MARK, 105, Harley Street, W. *Trans.* 1.

ELECTED

1893 HOWARD, R. J. BLISS, M.D., 31, Queen Anne Street, W.

1876 HOWSE, Sir HENRY GREENWAY, M.S., 59, Brook Street, W. (C. 1881–3, V.P. 1890–2.) *Trans.* 3.

1898 HULKE, SYDNEY BACKHOUSE, Ivy House, Walmer, Kent.

1897 HUMPHRYS, CHARLES B., Eagle House, Blandford, Dorset.

1897 HUNT, GEORGE BERTRAM, M.B., 47, Albemarle Crescent, Scarborough. *Trans.* 1, *C.C.* 1.

1892 HUNTER, WILLIAM, M.D., 103, Harley Street, W.

O.M. HUTCHINSON, JONATHAN, F.R.S., 15, Cavendish Square, W. (C. 1867–8, V.P. 1875–6.) *Trans.* 14, *C.C.* 1.

1896 HUTCHINSON, JONATHAN, jun., 15, Cavendish Square, W. (C. 1902– .) *Trans.* 4, *C.C.* 5.

1893 IONIDES, THEODORE HENRY, M.B., B.S., 30, Palmeira Square, West Brighton.

1883 JACKSON, GEORGE HENRY, Ashburton, Carew Road, Eastbourne.

O.M. JACKSON, J. HUGHLINGS, M.D., F.R.S., 3, Manchester Square, W. (C. 1872–3, V.P. 1887.) *Trans.* 2, *C.C.* 1.

1877 JACOBSON, WALTER HAMILTON ACLAND, M.B., M.Ch., 66, Great Cumberland Place, W. (C. 1890–2.) *Trans.* 2, *C.C.* 1.

1888 JAMES, JAMES THOMAS, M.D., 30, Harley Street, W.

1901 JOHNSON, EDWARD ANGAS, M.B., B.S., St. Catherine's, Prospect, South Australia.

1889 JOHNSON, RAYMOND, M.B., B.S., 11, Wimpole Street, W. (C. 1900– .) *Trans.* 5, *C.C.* 3.

1893 JOHNSTON, G. F., M.D., 3, Montagu Place, Montagu Square, W.

1878 JOHNSTON, WILLIAM, M.D., M.C., 16, Lonsdale Terrace, Upper Kent Street, Leicester.

1898 Jones, ROBERT, 11, Nelson Street, Liverpool. *Trans.* 2.

1872 JONES, THOMAS RIDGE, M.D., 4, Chesham Place, S.W. (C. 1892–3.)

1876 JORDAN, FURNEAUX, Harborne, Birmingham. *Trans.* 1.

1902 JOWERS, REGINALD F., 55, Brunswick Square, Brighton.

1886 JULER, HENRY EDWARD, 23, Cavendish Square, W.

1878 KEETLEY, CHARLES ROBERT BELL, 56, Grosvenor Street, W. (C. 1895–8.) *Trans.* 2.

1898 KELLOCK, THOMAS HERBERT, 8, Queen Anne Street, Cavendish Square, W. *Trans.* 2, *C.C.* 2.

O.M. KELLY, CHARLES, M.D., Ellesmere, Gratwicke Road, Worthing, Sussex.

1901 KELYNACK, THEO. N., M.D., 53, Harley Street, W.

1901 KEOGH, Lieut.-Col. ALFRED, C.B., M.D., 18, Victoria Street, S.W.

1897 KEYSER, CHARLES RALPH, 29, Hamilton Terrace, N.W.

1883 KIDD, PERCY, M.D., 60, Brook Street, W. (C. 1891–2, 1902– , S. 1899–1902.) *Trans.* 4, *C.C.* 9.

1887 Knaggs, R. LAWFORD, B.C., 27, Park Square, Leeds. *Trans.* 2.

ELECTED

1878 LACEY, THOMAS WARNER, 196, Burrage Road, Plumstead.

1897 LAMPLOUGH, CHARLES, Chatteris, Cambridgeshire.

1890 LANCASTER, ERNEST LE CRONIER, M.B., B.Ch., Winchester House, Swansea, S. Wales. *Trans.* 2.

1895 LANE, JAMES ERNEST, 46, Queen Anne Street, W. *Trans.* 1.

1883 LANE, WILLIAM ARBUTHNOT, M.B., M.S., 21, Cavendish Square, W. (C. 1893–6.) *Trans.* 29, *C.C.* 10.

O.M. LANGTON, JOHN, 62, Harley Street, W. (C. 1878–80, V.P. 1892–4, P. 1897–9.) *Trans.* 5.

1886 LANKESTER, HERBERT, M.D., Ingleborough, Woking, Surrey.

1885 LARDER, HERBERT, Whitechapel Infirmary, Vallance Road, E. *C.C.* 1.

1893 LAWSON, ARNOLD, M.D., 12, Harley Street, W.

O.M. LAWSON, GEORGE, 12, Harley Street, W. (S. 1871–3, C. 1874–6, V.P. 1881–3.) *Trans.* 16.

1900 LEAF, CECIL H., M.B., 75, Wimpole Street, Cavendish Square, W.

1877 Lediard, HENRY AMBROSE, M.D., 35, Lowther Street, Carlisle. (C. 1889, V.-P. 1902– .) *Trans.* 5, *C.C.* 1.

1896 Leech, PRIESTLEY, M.D., King Cross, Halifax.

1877 LEES, DAVID B., M.D., 22, Weymouth Street, W. (C. 1885, V.-P. 1902– .) *Trans.* 6.

1902 LEGG, THOMAS PERCY, M.B., 141, Harley Street, Cavendish Square, W.

1893 LENDON, EDWIN HARDING, M.D., 162, Holland Park Avenue, W.

1899 LESLIE, R. MURRAY, M.D., 26, Harley Street, Cavendish Square, W.

1901 LEWERS, ARTHUR HAMILTON NICHOLSON, M.D., 72, Harley Street, Cavendish Square, W.

1892 LEWIS, EDWARD JOHN, M.B., B.C., 74, Hamilton Terrace, N.W.

1895 LEWIS, ERNEST E., M.D., Springfield, Chelmsford.

1890 LITTLE, JOHN FLETCHER, M.B., 32, Harley Street, W. *C.C.* 5.

1891 LITTLEWOOD, HARRY, 25, Park Square, Leeds. *Trans.* 1.

1875 LIVEING, EDWARD, M.D., 52, Queen Anne Street, W.

1885 LOCKWOOD, CHARLES BARRETT, 19, Upper Berkeley Street, W. (C. 1896–9.) *Trans.* 7.

1876 LONGHURST, ARTHUR EDWIN TEMPLE, M.D., 26, Lower Belgrave Street, Eaton Square, S.W. (C. 1889–91.) *Trans.* 1.

1897 LOW, HAROLD, M.B., 10, Evelyn Gardens, South Kensington, S.W.

1899 LOW, V. WARREN, M.D., B.S., 27, Queen Anne Street, Cavendish Square, W.

1881 LUBBOCK, MONTAGU, M.D., 19, Grosvenor Street, W.

1876 LUCAS, R. CLEMENT, M.B., B.S., 50, Wimpole Street, W. (C. 1883–5, V.P. 1901– .) *Trans.* 11. *C.C.* 1.

1894 LUFF, ARTHUR PEARSON, M.D., 9, Queen Anne Street, Cavendish Square, W.

ELECTED

1879 LUNN, JOHN REUBEN, New Marylebone Infirmary, Rackham Street, Ladbroke Grove Road, W. (C. 1890–1.) *Trans.* 8, *C.C.* 16.

1893 LYS, HENRY GRABHAM, M.D., Southbrook, Suffolk Road, Bournemouth.

1889 MACBRIDE, P., M.D., 16, Chester Street, Edinburgh.

1891 MACDONALD, GREVILLE, M.D., 85, Harley Street, W.

1901 MCGAVIN, LAURIE HUGH, 21, Montagu Street, Portman Square, W. *C.C.* 1.

1881 MCHARDY, MALCOLM MACDONALD, 5, Savile Row, W. *Trans.* 1.

1882 MACKENZIE, FREDERIC MORELL, 29, Hans Place, S.W.

1891 MACKENZIE, HECTOR W. G., M.A., M.D., 34, Upper Brook Street, W. (C. 1902– .) *Trans.* 6, *C.C.* 1.

1879 MACKENZIE, STEPHEN, M.D., Merrycourt, Gt. Bookham, Leatherhead. (C. 1884, 1888–90, S. 1885–7. V.P. 1900–2.) *Trans.* 9, *C.C.* 10.

1884 MACKERN, JOHN, M.B., St. German's Lodge, Shooter's Hill Road, Blackheath, S.E.

1879 MACLAGAN, THOMAS JOHN, M.D., 9, Cadogan Place, S.W. (C. 1889–91.) *Trans.* 3.

1885 MACLAREN, RODERICK, M.D., Portland Square, Carlisle. *Trans.* 1.

1896 MAC RAE, FARQUHAR, M.B., 27A, Lowndes Street, Belgrave Square, S.W.

1898 MACREADY, JONATHAN F. C. H., 42, Devonshire Street, Portland Place, W.

1879 MAGILL, JAMES, M.D., M.C., Coldstream Guards, Queen Anne's Mansions, S.W.

1885 MAGUIRE, ROBERT, M.D., 4, Seymour Street, W. *Trans.* 1.

1881 MAKINS, GEORGE HENRY, C.B. 47, Charles Street, Berkeley Square, W. (C. 1892–4, 1898–1901, S. 1895–8.) *Trans.* 4, *C.C.* 7.

1887 MALCOLM, JOHN D., M.B., C.M., 13, Portman Street, W. (C. 1900– .) *Trans.* 1, *C.C.* 1.

1890 MANSON, PATRICK, C.M.G., M.D., C.M., F.R.S., 21, Queen Anne Street, W. (C. 1895–7.) *C.C.* 1.

1902 MARRIOTT, CECIL, M B., B.C., 5, Welford Road, Leicester.

1888 Marriott, HYDE, M.B., Dial House, Stockport.

1868 MARSH, F. HOWARD (*President*), 30, Bruton Street, W. (C. 1876–7, 1881–3, S. 1878–80, V.P. 1887–8, T. 1899–1901, P. 1901– .) *Trans.* 17, *C.C.* 7.

1887 MARTIN, SIDNEY, M.D., B.S., F.R.S., 10, Mansfield Street, W. (C. 1896–9.)

1888 MASON, DAVID JAMES, M.D., C.M., Rosemont, Maidenhead.

1892 MASTERS, JOHN ALFRED, M.D., 31, Albert Gate, Hyde Park, S.W.

1884 MAUDSLEY, HENRY CARR, M.D., 22, Collins Street, Melbourne, Victoria.

1897 MAY, CHICHESTER GOULD, M.D., 59, Cadogan Place, S.W.

1868 May, EDWARD HOOPER, M.D., High Cross, Tottenham, Middlesex.

1888 MAY, WILLIAM PAGE, M.D., B.Sc., 9, Manchester Square, W.; and Helouan, nr. Cairo, Egypt (October to April).

1901 MENZIES, F. U., Edinburgh University Union, Edinburgh.

ELECTED

1888 MENZIES, J. HERBERT, 47, Earl's Court Square, S.W.

1893 MERCER, WILLIAM BRACEWELL, M.B., B.C., Cliffe Cottage, Ripponden, near Halifax, Yorks.

1878 MEREDITH, WILLIAM APPLETON, C.M., 21, Manchester Square, W. (C. 1887–9.) *Trans.* 2.

1894 MICHELS, ERNST, M.D., 48, Finsbury Square, E.C.

1873 MICKLE, WILLIAM JULIUS, M.D., Grove Hall Asylum, Bow, E. (C. 1897–1900.)

1890 MILEY, MILES, M.A., M.B., 21, Belsize Avenue, Hampstead, N.W.

1896 MILLS-ROBERTS, ROBERT HERBERT, Llanberis, North Wales. *Trans.* 1.

1882 MONEY, ANGEL, M.D., Hunter Street, Sydney, New South Wales. (C. 1888–90.) *Trans.* 3.

1874 MORGAN, JOHN HAMMOND, C.V.O., 68, Grosvenor Street, W. (C. 1883–5, V.P. 1898–1901.) *Trans.* 2, *C.C.* 8.

1888 MORISON, ALEXANDER, M.D., 14, Upper Berkeley Street, W. *Trans.* 2, *C.C.* 1.

1899 MORISON, RUTHERFORD, 14, Saville Row, Newcastle-on-Tyne. *Trans.* 1.

1877 MORRIS, HENRY, M.A., M.B., 8, Cavendish Square, W. (C. 1884–6.) *Trans.* 9.

1877 MORRIS, MALCOLM ALEX., 8, Harley Street, W. (C. 1890–2.) *Trans.* 1, *C.C.* 1.

1901 MORSE, THOMAS HENRY, All Saints' Green, Norwich. *Trans.* 1.

1885 MOTT, FREDERICK WALKER, M.D., F.R.S., 25, Nottingham Place, W. (C. 1895–8.) *Trans.* 1, *C.C.* 4.

1899 MOUAT-BIGGS, CHARLES E. FORBES, 2, Fauconberg Villas, Bayshill, Cheltenham. *C.C.* 2.

1879 MOULLIN, CHARLES W. MANSELL, 69, Wimpole Street, W. (C. 1888–90.) *Trans.* 10, *C.C.* 4.

1899 MOYNIHAN, BERKELEY GEORGE ANDREW, M.S., 33, Park Square, Leeds. *Trans.* 1.

1875 MURPHY, SHIRLEY F., 22, Endsleigh Street, Tavistock Square, W.C. (C. 1888–90.) *C.C.* 1.

1885 MURRAY, ALEXANDER DALTON, M.B., Colombo, Ceylon.

1893 MURRAY, GEORGE REDMAYNE, M.D., 11, Ellison Place, Newcastle-on-Tyne.

1883 MURRAY, HUBERT MONTAGUE, M.D., 25, Manchester Square, W. (C. 1893–6.) *Trans.* 1.

1894 MURRAY, JOHN, 110, Harley Street, W. *C.C.* 1.

1872 MYRTLE, ANDREW S., M.D., 10, Park Parade, Harrogate. (C. 1892.)

1892 NASH, WALTER GIFFORD, 31, St. Peter's, Bedford.

1889 Newman, D., M.D., 18, Woodside Place, Glasgow. *Trans.* 4.

O.M. NORTON, ARTHUR TREHERN, C.B., Leyfields Wood, Ashampstead, Berks. (C. 1874–6.) *Trans.* 7.

O.M. NUNN, THOMAS WILLIAM, 8, Stratford Place, W. (C. 1873–4.) *Trans.* 9.

ELECTED

1880 O'CONNOR, BERNARD, M.D., 25, Hamilton Road, Ealing, W. *Trans.* 1.
1899 OGLE, CYRIL, M.B., 96, Gloucester Place, Portman Square, W. *Trans.* 1.
O.M. OGLE, JOHN WILLIAM, M.D., 96, Gloucester Place, Portman Square, W. (C. 1867–8, V.P. 1884–5.) *Trans.* 6.
1868 Ogle, WILLIAM, M.D., The Elms, Driffield Road, Derby.
1887 Oliver, THOMAS, M.D., 7, Ellison Place, Newcastle-upon-Tyne.
1887 OPENSHAW, THOMAS HORROCKS, M.B., 16, Wimpole Street, W. *C.C.* 1.
1863 OPPERT, FRANZ, M.D., 128, Leipzigerstrasse, Friedenau, Germany. *Trans.* 1.
1889 ORD, W. WALLIS, M.D., B.Ch., The Hall, Salisbury. *Trans.* 1, *C.C.* 1.
1887 ORMEROD, JOSEPH ARDERNE, M.D., 25, Upper Wimpole Street, W. (C. 1899–1902.) *C.C.* 2.
1884 ORMSBY, LAMBERT HEPENSTAL, M.D., 92, Merrion Square West, Dublin.
1883 ORTON, GEORGE HUNT, M.B., 1A, Campden Hill Road, Kensington, W.
1877 OWEN, Sir ISAMBARD, M.D., 40, Curzon Street, W. (C. 1888–90.) *Trans.* 4.
1888 OXLEY, ALFRED RICE, M.D., 7, Courtfield Road, South Kensington, S.W.
1888 PAGE, FREDERICK, M.D., 1, Saville Place, Newcastle-on-Tyne.
1875 PAGE, HERBERT W., M.C., M.B., 146, Harley Street, W. (C. 1882–4, V.P. 1899–1902.) *Trans.* 8, *C.C.* 2.
1884 PAGET, STEPHEN, 70, Harley Street, W. (C. 1898–1901.) *Trans.* 6, *C.C.* 2.
1881 PARKER, RUSHTON, M.B., B.S., 59, Rodney Street, Liverpool. (C. 1896–9.) *Trans.* 1, *C.C.* 2.
1890 PARKIN, ALFRED, M.S., 24, Albion Street, Hull. *Trans.* 2.
1894 PARKINSON, JOHN PORTER, M.D., 57, Wimpole street, W. *C.C.* 4.
1898 PARTRIDGE, ARTHUR A. H., M.D., M.Ch., St. Germans, Sutton, Surrey.
1881 PASTEUR, WILLIAM, M.D. (*Hon. Secretary*), 4, Chandos Street, W. (C. 1891–3, S. 1902– .) *Trans.* 5, *C.C.* 4.
1893 PATERSON, DONALD ROSE, M.D., C.M., 18, Windsor Place, Cardiff.
1900 PATON, EDWARD PERCY, M.D., 84, Park Street, Grosvenor Square, W *Trans.* 1.
1892 PAUL, FRANK THOMAS, 38, Rodney Street, Liverpool. *Trans.* 1.
1893 PAUL, JOHN ERNEST, M.B., 26, Queensborough Terrace, Kensington Gardens, W. *Trans.* 1.
O.M. PAVY, FREDERICK WILLIAM, M.D., F.R.S., 35, Grosvenor Street, W. (C. 1869–71, V.P. 1882–4.) *Trans.* 3.
1886 PAYNE, JOSEPH FRANK, M.D., 78, Wimpole Street, W. (C. 1897–1900.) *Trans.* 1.
1879 PEEL, ROBERT, 120, Collins Street East, Melbourne, Victoria.
1886 Penny, WILLIAM JOHN, Coombe, West Crewkerne, Somerset.
1887 PENROSE, FRANCIS GEORGE, M.D., 84, Wimpole Street, W. (C. 1896–9.) *Trans.* 1, *C.C.* 2.

ELECTED

1882 PEPPER, AUGUSTUS JOSEPH, M.S., M.B., 13, Wimpole Street, W. *Trans.* 1.

1900 PERKINS, GEORGE STEELE, M.D., 85, Wimpole Street, W.

1898 PERKINS, J. J., M.D., 41, Wimpole Street, Cavendish Square, W. *Trans.* 1, *C.C.* 1.

1895 PHEAR, ARTHUR G., M.D., B.C., 47, Weymouth Street, Portland Place, W. *Trans.* 2.

1895 PHILLIPS, F. B. WILLMER, M.B., 7, Harper Place, Bedford.

1884 PHILLIPS, SIDNEY, M.D., 62, Upper Berkeley Street, W. (C. 1893-6.) *Trans.* 7, *C.C.* 6.

O.M. PICK, THOMAS PICKERING, 18, Portman Street, W. (S. 1874–7, C. 1878–80, V.P. 1885–6.) *Trans.* 4.

1885 PITT, GEORGE NEWTON, M.D., 15, Portland Place, W. (C. 1894–6.) *Trans.* 6, *C.C.* 5.

1883 PITTS, BERNARD, M.A., M.C., 109, Harley Street, W. (C. 1893.) *Trans.* 5.

1871 Playne, ALFRED, M.B., Maidenhead.

1884 POLAND, JOHN, 2, Mansfield Street, Cavendish Square, W.

1884 POLLARD, BILTON, B.S., 24, Harley Street, W. (C. 1895–8.) *Trans.* 2.

1868 POLLOCK, JAMES EDWARD, M.D., 37, Collingham Place, S.W. (C. 1878–80.)

1871 POORE, GEORGE VIVIAN, M.D., 24A, Portland Place, W. (C. 1879–81, V. P. 1901– .) *Trans.* 4.

1881 POWELL, H. A., M.A., 44, Sandgate Road, Folkestone.

O.M. POWELL, Sir R. DOUGLAS, Bart., K.C.V.O., M.D., 62, Wimpole Street, W. (C. 1874–6, V.P. 1889–90, P. 1899-1901.) *Trans.* 4.

1868 PRENTIS, CHARLES, Surgeon-Major, Bengal Medical Service; India.

1902 PRICE, F. W., M.B., 4, Endsleigh Gardens, N.W.

1884 PRINGLE, JOHN JAMES, M.B., 23, Lower Seymour Street, W. (C. 1890-2, 1897–1900.) *Trans.* 1, *C.C.* 3.

1884 PYE-SMITH, PHILIP HENRY, M.D., F.R.S., 48, Brook Street, W. (C. 1890–2.) *Trans.* 1.

1896 PYE-SMITH, RUTHERFORD JOHN, 350, Glossop Road, Sheffield.

1893 RAKE, ALFRED THEODORE, M.B., B.S., 8, Sheriff Road, West Hampstead, N.W.

1895 RAMSAY, HERBERT MURRAY, 35A, Hertford Street, Mayfair, W.

1889 RANKING, JOHN E., M.D., Hanover House, Tunbridge Wells.

1883 READ, THOMAS LAURENCE, 11, Petersham Terrace, Queen's Gate, S.W.

1902 REID, Sir JAMES, Bart., G.C.V.O., K.C.B., 72, Grosvenor Street, Grosvenor Square, W.

1901 REISSMANN, CHARLES, M.B., Knutsford, Glenelg, South Australia.

1889 Rennie, GEORGE EDWARD, M.D., College Street, Hyde Park, Sydney, N.S.W.

1868 RICE, MICHAEL W., M.D., Elmbank, Hargrave, Northamptonshire, (C. 1876–8.)

ELECTED

1898 RICHARDS, JOSEPH STEWART, M.D., B.S., The Infirmary, East Dulwich, S.E. *C.C.* 2.

O.M. RINGER, SYDNEY, M.D., F.R.S., 15, Cavendish Place, W. (C. 1871–2.) *Trans.* 1.

1896 RISDON, WILLIAM ELLIOT, M.D., 81, Chancery Lane, W.C.

1873 **Roberts**, DAVID LLOYD, M.D., 11, St. John Street, Manchester.

1888 ROBERTS, FRANK ERNEST, Tulse Dale Villa, Lower Norwood, S.E.

1883 ROBERTS, FREDERICK THOMAS, M.D., 102, Harley Street, W. (C. 1892–4.)

1900 ROBERTS, GRIFFITH LLOYD, M.B., C.M., Castle Square, Carnarvon, North Wales.

1890 ROBERTSON, ROBERT, M.D., Belgrave Road, Ventnor, Isle of Wight.

1885 ROBINSON, ARTHUR HENRY, M.D., St. Mary's Infirmary, Highgate Hill, N. *C.C.* 3.

1890 ROBINSON, GEORGE SOMERVILLE, Surgeon-Major, 13, Lupus Street, St. George's Square, S.W.

1892 ROBINSON, HENRY BETHAM, M.D., M.S., 1, Upper Wimpole Street, W. (C. 1901–). *Trans.* 6, *C.C.* 9.

1885 ROBSON, A. W. MAYO, 8, Park Crescent, W., and 7, Park Square, Leeds. (C. 1893–6.) *Trans.* 14.

1897 ROGER-SMITH, HUGH, M.D., 1, College Terrace, Fitzjohn's Avenue, N.W. *Trans.* 1.

1889 ROLLESTON, HUMPHRY DAVY, M.A., M.D., 55, Upper Brook Street, Grosvenor Square, W. (C. 1899–1902.) *Trans.* 9. *C.C.* 7.

1888 ROPER, ARTHUR, "Colby," Lewisham Hill, Blackheath. *Trans.* 1.

1897 ROSS, FREDERICK WILLIAM FORBES, M.D., 15, Gower Street, W.C.

1877 **ROTH**, BERNARD, 38, Harley Street, Cavendish Square, and "Wayside," Preston Park Avenue, Brighton. *Trans.* 1, *C.C.* 4.

1890 ROUGHTON, EDMUND WILKINSON, B.S., 38, Queen Anne Street, W. *C.C.* 1.

1874 ROWLAND, EDWARD ROGER, Dordrecht, Wodehouse, S. Africa.

1898 RUSSELL, ALFRED ERNEST, M.D., B.S., Melton House, Manor Park, Lee, S.E.

1900 RUSSELL, J. S. RISIEN, M.D., 44, Wimpole Street, Cavendish Square, W. *Trans.* 1, *C.C.* 1.

1887 RUTHERFOORD, H. T., M.B., Salisbury House, Taunton.

1885 RYLE, REGINALD JOHN, M.D., Green View, Hadley Green, Barnet.

1882 SAINSBURY, HARRINGTON, M.D., 52, Wimpole Street, W. (C. 1898–1901.)

1899 SANDIFER, HENRY STEPHEN, M.D., B.S., 11, Porchester Gardens, W.

1893 SANSOM, ARTHUR ERNEST, M.D., 84, Harley Street, W. *Trans.* 1, *C.C.* 1.

1873 SAVAGE, GEORGE HENRY, M.D., 3, Henrietta Street, W. (C. 1882–3.)

1886 SAVILL, THOMAS DIXON, M.D., 60, Upper Berkeley Street, W. *Trans.* 1, *C.C.* 3.

1899 SCHORSTEIN, GUSTAVE, M.B., 11, Portland Place, W.

1886 SCOTT, ALFRED, 15, German Place, Brighton.

ELECTED

1894 SCOTT, BERNARD, "Hartington," Poole Road, Bournemouth.
1900 SCOTT, KENNETH, M.B., 7, Manchester Square, W.
1892 SCOTT, RICHARD JAMES HERBERT, 28, Circus, Bath. *C.C.* 1.
1902 SEARS, ALFRED ERNEST, 33, Lee Terrace, Blackheath, S.E. *Trans.* 1.
1869 SEDGWICK, LEONARD WILLIAM, M.D., 69, Gloucester Terrace, Hyde Park, W. (C. 1879–81, V.P. 1897–1900.)
1897 SEGUNDO, CHARLES SEMPILL DE, M.B., B.S., 6, Brook Street, Hanover Square, W.
1892 SELWYN-HARVEY, JOHN STEPHENSON, M.D., 1, Astwood Road, S.W.
1878 SEMON, Sir FELIX, M.D., 39, Wimpole Street, W. (C. 1885–7.) *Trans.* 8, *C.C.* 3.
1884 SHARKEY, SEYMOUR J., M.D., 22, Harley Street, W. (C. 1895–8.)
1898 SHAW, HAROLD BATTY, M.D., 7, Devonshire Street, Portland Place, W. *C.C.* 3.
1889 SHAW, LAURISTON ELGIE, M.D., 64, Harley Street, W. *C.C.* 1.
1875 SHERWOOD, ARTHUR PAUL, 8, Seaside Road, Eastbourne.
1886 SILCOCK, ARTHUR QUARRY, M.D., B.S., 52, Harley Street, W. (C. 1896–9.) *Trans.* 5. *C.C.* 2.
1879 SKERRITT, EDWARD MARKHAM, M.D., Edgecumbe House, Richmond Hill, Clifton, Bristol. (C. 1895–8.) *Trans.* 2.
1872 SLIGHT, GEORGE, M.D., 14, Old Burlington Street, W.
1882 SMITH, E. NOBLE, 24, Queen Anne Street, W. *Trans.* 1.
1896 SMITH, EBENEZER STANLEY, M.D., 68, Wimpole Street, Cavendish Square, W.
1888 SMITH, FREDERICK J., M.D., 138, Harley Street, W. *Trans.* 2.
1884 SMITH, R. PERCY, M.D., 36, Queen Anne Street, Cavendish Square, W. (C. 1898–1901.)
1893 SMITH, SOLOMON CHARLES, M.D., Four Oaks, Walton-on-Thames.
O.M. SMITH, Sir THOMAS, Bart., 5, Stratford Place, W. (C. 1869–71, V.P. 1880–2.) *Trans.* 14.
1894 SMITH, THOMAS RUDOLPH, M.B., B.C., 25, Bridge Road, Stockton-on-Tees.
1893 SNAPE, ERNEST ALFRED, M.D., 39, Welbeck Street, W.
1902 SNELL, SIDNEY HERBERT, M.D., B.S., D.P.H., 27, New Cavendish Street, W.
1868 SNOW, WILLIAM V., M.D., Richmond Gardens, Bournemouth.
1890 SOLLY, ERNEST, M.B., Strathlea, Harrogate, Yorks. *C.C.* 1.
1888 SPENCER, WALTER GEORGE, M.S., M.B., 35, Brook Street, W. (C. 1896–9.) *Trans.* 11, *C.C.* 11.
1885 SPICER, FREDERICK, M.D., 17, Wimpole Street, Cavendish Square, W.
1888 SPICER, ROBERT HENRY SCANES, M.D., 28, Welbeck Street, W. *C.C.* 1.
1882 SPOONER, FREDERICK HENRY, M.D., 4, Maitland Place, Lower Clapton, N.E.

ELECTED

1896 SPURRELL, CHARLES, Medical Superintendent, Poplar and Stepney Sick Asylum, Devon's Road, Bromley-by-Bow, E.

1876 SQUIRE, A. BALMANNO, 24, Weymouth Street, W. *Trans.* 5, *C.C.* 4.

1892 STABB, EWEN CARTHEW, 57, Queen Anne Street, W. *C.C.* 1.

1879 STAPLES, FRANCIS PATRICK, Brigade-Surgeon, Army (retired). (Address uncommunicated.)

1896 STEWARD, FRANCIS JAMES, M.B., B.S., 24, St. Thomas's Street, London Bridge, S.E. *Trans.* 2.

1874 **Stirling,** EDWARD C., M.D. [care of Messrs. ELDER & Co., 7, St. Helen's Place, E.C.], Adelaide, South Australia.

1888 STOKER, GEORGE, 14, Hertford Street, W. *C.C.* 2.

1899 STONE, WILLIAM GREAM, M.B., 93, Denmark Hill, S.E.

1884 STONHAM, CHARLES, 4, Harley Street, W. *C.C.* 3.

1878 STRUGNELL, FREDERICK WILLIAM, Grove End House, Highgate Road, Highgate, N.W. *C.C.* 1.

1878 **Sturge,** WILLIAM ALLEN, M.D., 29, Boulevard Dubouchage, Nice, France.

1894 SUTHERLAND, GEORGE A., M.D., 73, Wimpole Street, Cavendish Square, W. *Trans.* 1.

1882 SYMONDS, CHARTERS JAMES, M.S., 58, Portland Place, W. (C. 1888–9, 1901– , S. 1898–1901.) *Trans.* 12, *C.C.* 16.

1876 SYMONDS, HORATIO PERCY, 35, Beaumont Street, Oxford.

1885 TAIT, EDWARD SABINE, M.D., 48, Highbury Park, N.

1885 TAIT, HENRY BREWER, Lincluden, Sunnyside Road, Hornsey Lane, N.

1891 TATE, WALTER WILLIAM HUNT, M.D., 32, Queen Anne Street, W.

1886 TAY, WAREN, 4, Finsbury Square, E.C.

1878 TAYLER, FRANCIS THOMAS, M.B., 224, Lewisham High Road, S.E.

1897 TAYLOR, E. CLAUDE, M.D., B.S., Eland House, Rosslyn Hill, Hampstead, N.W.

1875 **TAYLOR,** FREDERICK, M.D., (*Treasurer*), 20, Wimpole Street, W. (S. 1879–81, C. 1882–4, V.P. 1894–7, T. 1901– .) *Trans.* 13, *C.C.* 3.

1889 **Taylor,** HENRY HERBERT, 10, Brunswick Place, Hove, Sussex.

1890 TAYLOR, JAMES, M.D., 49, Welbeck Street, W. *C.C.* 3.

1882 TAYLOR, SEYMOUR, M.D., 16, Seymour Street, W. *Trans.* 1, *C.C.* 3.

1885 **Taylor,** W. C. EVERLEY, 34, Queen Street, Scarborough.

1886 TEALE, THOMAS PRIDGIN, M.B., F.R.S., 38, Cookridge Street, Leeds. (C. 1897–1900.)

1896 TEMPLETON, GEORGE, M.B., 10, Seymour Street, Portman Square, W.

1890 THANE, EDGAR HERBERT, M.D., Wagga-Wagga, New South Wales.

1882 THIN, GEORGE, M.D., 63, Harley Street, W. *Trans.* 1.

1886 THOMPSON, CHARLES HERBERT, M.D., 133, Harley Street, Cavendish Square, W.

O.M. THOMPSON, EDMUND SYMES, M.D., 33, Cavendish Square, W. (C. 1880–2.) *Trans.* 1.

ELECTED

O.M. THOMPSON, Sir HENRY, Bart., 35, Wimpole Street, W. (C. 1867–8, V.P. 1886–7.) *Trans.* 3.

1897 THOMSON, H. CAMPBELL, M.D., 34, Queen Anne Street, Cavendish Square, W. *C.C.* 1.

1894 THOMSON, STCLAIR, M.D., 28, Queen Anne Street, W. *Trans.* 2, *C.C.* 8.

1896 THORNE, WILLIAM BEZLY, 53, Upper Brook Street, W. *C.C.* 2.

1887 THORNTON, JOHN KNOWSLEY, M.B., C.M., Hildersham Hall, Cambridge. (C. 1890–1.)

1872 THORNTON, WILLIAM PUGIN, 35, St. George's Road, Canterbury, *Trans.* 5.

1885 THURSFIELD, THOMAS WILLIAM, M.D., Selwood, Beauchamp Square, Leamington.

1899 TILLEY, HERBERT, M.D., 89, Harley Street, Cavendish Square, W. *C.C.* 1.

1891 TOMSON, W. BOLTON, M.D., Park Street West, Luton, Bedfordshire.

1897 TOOGOOD, F. SHERMAN, M.D., The Infirmary, 282, High Street, Lewisham, S.E.

1892 TOOTH, HOWARD HENRY, C.M.G., M.D., 34, Harley Street, W. *Trans.* 1.

1887 TOTSUKA, KANKAI, Tokio, Japan.

1874 TRAVERS, WILLIAM, M.D., 2, Phillimore Gardens, Kensington, W.

1884 TREVES, Sir FREDERICK, Bart., C.B., K.C.V.O., 6, Wimpole Street, W. (C. 1893.) *Trans.* 7.

1897 TUBBY, ALFRED HERBERT, M.S., 25, Weymouth Street, Portland Place, W. *Trans.* 2, *C.C.* 3.

1899 TULLIS, GEORGE AUGUSTUS, M.D., Holmbush, Grove Road, Southsea.

1882 TURNER, GEORGE ROBERTSON, 41, Half Moon Street, W. (C. 1897–1900.) *Trans.* 9. *C.C.* 6.

1888 TURNER, PHILIP DYMOCK, M.D., Sudbury, Isle of Wight. *Trans.* 1.

1898 TURNER, WILLIAM, M.B., 53, Queen Anne Street, Cavendish Square, W.

1893 TURNEY, HORACE GEORGE, M.D., 68, Portland Place, W. *C.C.* 4.

1878 TYSON, WILLIAM JOSEPH, M.D., 10, Langhorne Gardens, Folkestone. (C. 1886–8, V.-P. 1900–2.) *Trans.* 8.

1881 UHTHOFF, JOHN CALDWELL, M.D., Wavertree House, Brunswick Place, Brighton. (C. 1898–1901.)

1868 VENNING, EDGCOMBE, 30, Cadogan Place, S.W. (C. 1876–8.) *Trans.* 2.

1890 VOELCKER, ARTHUR FRANCIS, M.D., B.S., 101, Harley Street, W. (C. 1900– .) *Trans.* 2, *C.C.* 2.

1886 WADE, CHARLES H., 19, Royal Terrace, Southend-on-Sea.

1868 WAGSTAFFE, WILLIAM WARWICK, Purleigh, St. John's Hill, Sevenoaks. (C. 1878.)

1886 **WAINEWRIGHT**, BENJAMIN, M.B., C.M., 47, Weymouth Street, W. *Trans.* 2, *C.C.* 2.

1885 WAKLEY, THOMAS, jun., 5, Queen's Gate, S.W.

1885 WALKER, CHARLES ROTHERHAM, M.D., 15, Sackville Road, Hove, Sussex.

1899 WALKER, HENRY ROE, 8, Harley Street, W.

ELECTED

1896 WALLACE, CUTHBERT SYDNEY, 26, Upper Wimpole Street, W. (C. 1902– .) *Trans.* 5, *C.C.* 3.

1890 WALLIS, FREDERICK CHARLES, M.B., B.C., 107, Harley Street, W. (C. 1902– .) *Trans.* 4, *C.C.* 10.

1875 **WALSHAM**, WILLIAM J., C.M., 77, Harley Street, W. (C. 1882–4, V.P. 1896–9.) *Trans.* 5, *C.C.* 1.

1888 WALTERS, FREDERICK RUFENACHT, M.D., Crooksbury Sanatorium, Farnham, Surrey. *C.C.* 2.

1896 WARD, EDWARD, M.B., 22, Park Place, Leeds. *Trans.* 1.

1888 Warner, PERCY, Rydal, Woodford Green, Essex.

1891 WATERHOUSE, HERBERT FURNIVALL, M.D., C.M., 81, Wimpole Street, W.

1896 **WEBER**, FRED. PARKES, M.D., 19, Harley Street, W. *C.C.* 5.

O.M. WEBER, Sir HERMANN D., M.D., 10, Grosvenor Street, W. (C. 1867–71, V.P. 1873–5.) *Trans.* 9.

1882 WEST, SAMUEL, M.D., 15, Wimpole Street, W. (C. 1887–9.) *Trans.* 16, *C.C.* 5.

1895 WETHERED, FRANK J., M.D., 83, Harley Street, W.

1874 WHEELHOUSE, CLAUDIUS GALEN, Hilary Place, Leeds. *Trans.* 1.

1868 WHIPHAM, THOMAS TILLYER, M.D., 11, Grosvenor Street, W. (C. 1878–80, V.P. 1894–7.) *Trans.* 13.

1891 WHITE, CHARLES PERCIVAL, M.B., B.C., 22, Cadogan Gardens, S.W.

1882 WHITE, EDWIN FRANCIS, Westlands, 280, Upper Richmond Road, Putney, S.W.

1890 WHITE, GILBERT B. MOWER, M.B., B.S., 112, Harley Street, W.

1883 WHITE, WILLIAM HALE, M.D. 65, Harley Street, W. (C. 1887–9, 1896–9, S. June, 1893–6.) *Trans.* 16, *C.C.* 4.

1883 WHITE, WILLIAM HENRY, M.D., 43, Weymouth Street, W. *Trans.* 1. *C.C.* 1.

1894 WHITELOCKE, RICHARD HENRY ANGLIN, M.B., C.M., 6, Banbury Road, Oxford.

1897 WHITFIELD, ARTHUR, M.D., 12, Upper Berkeley Street, Portman Square, W.

1897 WHITING, ARTHUR JOHN, M.D., 142, Harley Street, W.

1882 WHITTLE, EDWARD GEORGE, M.D., 65, Dyke Road, Brighton.

1871 WIGHT, GEORGE, M.B., C.M., 428, Liverpool Road, N.

1879 WILCOX, HENRY, M.B., Newlyn, Fleet, Hants.

1894 **WILKIN**, GRIFFITH CHARLES, West Coker, near Yeovil, Somerset.

1884 WILLCOCKS, FREDERICK, M.D., 14, Mandeville Place, W. *C.C.* 1.

O.M. WILLETT, ALFRED, 36, Wimpole Street, W. (C. 1872–5, V.P. 1889–91.) *C.C.* 2.

1890 WILLETT, EDGAR, M.B., 22, Queen Anne Street, Cavendish Square, W.

1898 WILLEY, FREDERICK INGOR, M.B., B.S., The Wych, 8, Avenue Road, Highgate, N.

ELECTED

1888 WILLIAMS, CAMPBELL, 18, Queen Anne Street, Cavendish Square, W. *Trans.* 2.

O.M. WILLIAMS, CHARLES THEODORE, M.D., 2, Upper Brook Street, W. (C. 1877–9, V.P. 1889–90.) *Trans.* 8.

1888 WILLIAMS, DAWSON, M.D., 2, Wyndham Place, Bryanston Square, W. (C. 1893–7.)

1881 WILLIAMS, Sir JOHN, Bart., M.D., 63, Brook Street, W. (C. 1885–6.)

1890 WILLIAMS, W. ROGER, Beaufort House, Clifton Down, Clifton, Bristol.

O.M. WILLIS, FRANCIS, M.D., The Spa, Braceborough, Stamford.

1893 WILLS, JOSEPH PEARCE BUDGETT, M.D., Bexhill, Hastings.

1889 WILLS, WILLIAM ALFRED, M.D., 29, Lower Seymour Street, W.

1886 WILSON, ALBERT, M.D., Minto House, South Woodford, Essex. *Trans.* 1, *C.C.* 2.

1888 Wilson, CLAUDE, M.D., C.M., Belmont, Tunbridge Wells. *Trans.* 2.

1891 WILSON, THEODORE STACEY, M.D., C.M., 29, Temple Row, Birmingham. *Trans.* 2.

1890 WOOD, NEVILLE, 42, Elvaston Place, Queen's Gate, S.W.

1883 WOODCOCK, JOHN ROSTRON, Abberley, near Stourport, Worcestershire.

1879 WOODWARD, GEORGE P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.

1894 WOOLLETT, CHARLES JEROME, "Ambleside," Streatham, S.W.

1884 WORTS, EDWIN, 6, Trinity Street, Colchester.

1892 WYNTER, WALTER ESSEX, M.D., B.S., 30, Upper Berkeley Street, W. *C.C.* 4.

1872 YEO, I. BURNEY, M.D., 44, Hertford Street, W. (C. 1881–3.) *Trans.* 8.

[*It is requested that any change of Title or Residence be communicated to the Secretaries before the* 1*st of July in each year, in order that the list may be made as correct as possible.*]

LIST OF MEMBERS.

ORIGINAL MEMBERS (ALPHABETICALLY).

Henry Arnott.
Richard Barwell.
Henry Charlton Bastian, M.D., F.R.S.
Sir Wm. Henry Broadbent, Bart., K.C.V.O., M.D, F.R.S.
Thomas Bryant.
Thomas Buzzard, M.D.
William Cayley, M.D.
Sir William Selby Church, Bart., K.C.B., M.D.
Edward Clapton, M.D.
John Couper.
William Howship Dickinson, M.D.
Sir Dyce Duckworth, M.D.
Christopher Heath.
Timothy Holmes.
Jonathan Hutchinson, F.R.S.
J. Hughlings Jackson, M.D., F.R.S.
Charles Kelly, M.D.
John Langton.
George Lawson.
Arthur Trehern Norton, C.B.
Thomas William Nunn.
John William Ogle, M.D.
Frederick William Pavy, M.D., F.R.S.
Thomas Pickering Pick.
Sir Richard Douglas Powell, Bart., M.D., K.C.V.O.
Sydney Ringer, M.D., F.R.S.
Sir Thomas Smith, Bart.
Edmund Symes Thompson, M.D.
Sir Henry Thompson, Bart.
Sir Hermann D. Weber, M.D.
Alfred Willett.
Charles Theodore Williams, M.D.
Francis Willis, M.D.

ARRANGED ACCORDING TO DATE OF ELECTION.

1868 Constantine Holman, M.D.
Thomas Tillyer Whipham, M.D.
Christian G. H. Bäumler, M.D.
James Grey Glover, M.D.
T. Henry Green, M.D.
Howard Marsh.
Charles Prentis.
Edgcombe Venning.

1868 John Ford Anderson, M.D.
George Granville Bantock, M.D.
George Charles Bright, M.D.
Frank W. Cooper.
Julian Evans, M.B.
Edward Hooper May, M.D.
William Warwick Wagstaffe.
William Ogle, M.D.

1868 James Edward Pollock, M.D.
Franz Oppert, M.D.
William V. Snow, M.D.
Charles Drage, M.D.
Frederick Royston Fairbank, M.D.
Michael W. Rice, M.D.
1869 Leonard William Sedgwick, M.D.
J. Warrington Haward.
Henry Frederick Augustus Goodridge, M.D.
Olliver Thomas Duke, M.B.
1871 Alfred Playne, M.B.
George Wight, M.B.
Ebenezer Diver, M.D.
George Vivian Poore, M.D.
1872 I. Burney Yeo, M.D.
Henry Harris, M.D.
William Pugin Thornton.
Sir G. Anderson Critchett.
J. C. J. Fenwick, M.D.
Andrew J. Myrtle, M.D.
Sir William Bartlett Dalby.
Thomas Ridge Jones, M.D.
George Slight, M.D.
1873 William Julius Mickle, M.D.
David Lloyd Roberts, M.D.
George Henry Savage, M.D.
Edwin Chisholm, M.D.
Thomas Churton, M.D.
1874 John Hammond Morgan, C.V.O.
Edward R. Rowland.
Claudius Galen Wheelhouse.
Edward C. Stirling, M.D.
Sir William Henry Bennett, K.C.V.O.
William Travers, M.D.
William Brown Holderness.
Andrew Clark.
1875 Sir Thomas Barlow, Bart., K.C.V.O., M.D.
Sidney Coupland, M.D.
Clinton T. Dent.
C. D. B. Hale.
Edward Liveing, M.D.
William J. Walsham, C.M.
Rickman John Godlee, M.S.
Arthur Paul Sherwood.
T. Gilbart-Smith, M.D.
James Frederic Goodhart, M.D.
Sir William Richard Gowers, M.D., F.R.S.
William Smith Greenfield, M.D.
Shirley F. Murphy.
1875 Herbert W. Page.
Frederick Taylor, M.D.
1876 Arthur E. J. Barker.
Horatio Percy Symonds.
A. Balmanno Squire.
David White Finlay, M.D.
Sir Henry Greenway Howse, M.S.
Furneaux Jordan.
R. Clement Lucas, B.S.
George Buckston Browne.
Arthur Edwin Temple Longhurst, M.D.
1877 Robert Hogarth Clay, M.D.
A. Pearce Gould, M.S.
Henry Radcliffe Crocker, M.D.
David B. Lees, M.D.
Walter Hamilton Acland Jacobson, M.B., M.Ch.
Sir Isambard Owen, M.D.
William Ewart, M.D.
Henry Morris, M.B.
Henry Ambrose Lediard, M.D.
Bernard Roth.
Henry Hugh Clutton.
Malcolm Alex. Morris.
1878 George P. Field.
Thomas Warner Lacey.
Thomas Colcott Fox, M.B.
Sir Felix Semon, M.D.
Henry de Fonmartin, M.D.
C. H. Golding-Bird, M.B.
Donald William Charles Hood, M.D.
Lord Lister, F.R.S.
Francis Thomas Tayler, M.B.
F. de Havilland Hall, M.D.
William Allen Sturge, M.D.
William Joseph Tyson, M.D.
William Johnston, M.D.
Charles Robert Bell Keetley.
William Appleton Meredith, C.M.
1879 Frederick William Strugnell.
William Edward Burton.
James Magill, M.D.
Edward Markham Skerritt, M.D.
Henry Wilcox, M.B.
John Abercrombie, M.D.
Stephen Mackenzie, M.D.
William Harrison Cripps.
Francis Patrick Staples.
Geo. Courtenay Henderson, M.D.
Thomas John Maclagan, M.D.
Henry Davy.

1879 Thos. Walter Harropp Garstang.
Charles W. Mansell Moullin.
John Reuben Lunn.
George P. M. Woodward, M.D.
Robert Peel.
Frederic S. Dennis, M.D.
1880 T. Mark Hovell.
Wyndham Cottle, M.D.
Henry Francis Baker.
Bernard O'Connor, M.D.
Charles Edward Beevor, M.D.
1881 George Henry Makins, C.B.
Robert William Burnet, M.D.
James Kingston Fowler, M.D.
Charles Edward Harrison, M.B.
Malcolm Macdonald McHardy.
Rushton Parker.
Sir John Williams, Bart, M.D.
Montagu Lubbock, M.D.
William Pasteur, M.D.
John Caldwell Uhthoff, M.D.
Henry Treutham Butlin.
H. A. Powell, M.A.
1882 George Robertson Turner.
E. Noble Smith.
Robert William Goldie.
Frederick Charles Barker, M.D.
Frederic Morell Mackenzie.
Daniel Colquhoun, M.D.
Seymour Taylor, M.D.
Philip Henry Bindley, M.B.
Edward George Whittle, M.D.
D. H. Goodsall.
Frederick Henry Spooner, M.D.
Frederick Haycraft Berry, M.D.
Herbert Collier, M.D.
Samuel West, M.D.
Emile Emond, M.D.
Charters James Symonds, M.S.
Angel Money, M.D.
C. F. Coxwell, M.B.
Augustus Joseph Pepper, M.B.
Harrington Sainsbury, M.D.
George Thin, M.D.
Edwin Francis White.
1883 Anthony A. Bowlby, C.M.G.
Percy Kidd, M.D.
William Henry White, M.D.
Hubert Montague Murray, M.D.
Robert Fitzroy Benham.
William Henry Allchin, M.D.
John Mitchell Bruce, M.D.
William Arbuthnot Lane, M.S.
Bernard Pitts.
1883 William Hale White, M.D.
William Coode Adams, M.B.
Robert Leamon Bowles, M.D.
George Henry Jackson.
George Hunt Orton, M.B.
Thomas Laurence Read.
Frederick Thomas Roberts, M.D.
Charles Alfred Ballance, M.S.
John Rostron Woodcock.
1884 Frederick Willcocks, M.D.
R. Percy Smith, M.D.
Edgar Duke.
John Mackern, M.B.
Paul M. Chapman, M.D.
Wilmot Parker Herringham, M.D.
Philip Henry Pye-Smith, M.D. F.R.S.
Charles Stonham.
Dudley Wilmot Buxton, M.D.
Edwin Worts.
Seymour J. Sharkey, M.D.
Sir Frederick Treves, Bart., C.B., K.C.V.O.
John James Pringle, M.B.
Frederick Lucas Benham, M.D.
Walter Edmunds, M.C.
Stephen Paget.
Lambert Hepenstal Ormsby, M.D.
John Poland.
Edwin Leonard Adeney, M.D.
Sir Victor Horsley, F.R.S.
Henry Carr Maudsley, M.D.
Bilton Pollard, B.S.
1885 Frederick Spicer, M.B.
Herbert Larder.
James Berry, B.S.
Fred. Walker Mott, M.D., F.R.S.
George Newton Pitt, M.D.
W. C. Everley Taylor.
Sidney Phillips, M.D.
A. W. Mayo Robson.
Thomas Wakley, jun.
Herbert William Allingham.
Thomas William Thursfield, M.D.
Alexander Dalton Murray, M.B.
Robert Maguire, M.D.
Robert Alexander Gibbons, M.D.
Henry Brewer Tait.
Charles Rotherham Walker, M.D.
Richard Caton, M.D.
Arthur Henry Robinson, M.D.
Edward Sabine Tait, M.B.
William Bruce Clarke, M.B.
Charles Barrett Lockwood.

1885 Reginald J. Ryle, M.D.
J. Michell Clarke, M.B.
Henry George Armstrong.
Roderick Maclaren, M.D.
W. Watson Cheyne, F.R.S.
Edward Liveing Fenn, M.D.
1886 Thomas Dixon Savill, M.D.
Charles Henry Wade.
Benjamin Wainewright, M.B.
Waren Tay.
William John Penny.
William Henry Battle.
James Hardie, M.D.
Francis Henry Hawkins, M.D.
R. Hingston Fox, M.D.
Henry Edward Juler.
John Ward Cousins, M.D.
Joseph Frank Payne, M.D.
T. Pridgin Teale, F.R.S.
H. Lankester, M.D.
Arthur T. Davies, M.D.
Charles Herbert Thompson, M.D.
Arthur Quarry Silcock, M.D., B.S.
Henry Handford, M.D.
Alfred Scott.
Albert Wilson, M.D.
1887 Archibald E. Garrod, M.D.
H. T. Rutherfoord, M.B.
Kankai Totsuka.
Thomas Oliver, M.D.
Francis George Penrose, M.D.
Samuel Herbert Habershon, M.D.
John Knowsley Thornton.
Oswald Auchinleck Browne, M.D.
Joseph Arderne Ormerod, M.D.
J. H. E. Brock, M.B., B.S.
Francis William Clark.
A. H. Weiss Clemow, M.D., C.M.
E. Hurry Fenwick.
R. Lawford Knaggs, B.C.
John D. Malcolm, M.B., C.M.
Sidney Martin, M.D., B.S., F.R.S.
Thomas Horrocks Openshaw, M.B.
1888 A. G. Barrs, M.D.
J. W. Batterham, M.B., B.S.
Montagu Handfield-Jones, M.D.
Alfred Rice Oxley, M.D.
Arthur Roper, M.D.
Robert Henry Scanes Spicer, M.D.
Campbell Williams.
Frederic S. Eve.
Alexander Morison, M.D.
Frederick Page, M.D.
Frederick J. Smith, M.D.

1888 Frederick R. Walters, M.D.
Claude Wilson, M.D., C.M.
Charles H. Gage-Brown, M.D.
J. H. Menzies.
Frank Ernest Roberts.
George Stoker.
Robert Ashton Bostock.
Hugh Armstrong.
Hyde Marriott, M.B.
Percy Warner.
J. T. James, M.D.
Edwin A. Barton.
W. Page May, M.D.
Philip D. Turner, M.D.
Dawson Williams, M.D.
Augustus W. Addinsell, M.B., C.M.
John Anderson, M.D.
Henry French Banham, M.D.
George Haynes Hetherington.
David James Mason, M.D., C.M.
Walter G. Spencer, M.B., M.S.
1889 Theodore Dyke Acland, M.D.
Raymond Johnson, M.B., B.S.
H. D. Rolleston, M.A., M.D.
P. MacBride, M.D.
D. Newman, M.D.
Herbert Elwin Harris, M.B.
John E. Ranking, M.D.
William Alfred Wills, M.D.
Edward Ashby Fardon.
Stanley Boyd, B.S.
George Ezra Halstead, M.D., B.S.
Henry Herbert Taylor.
John Duncan, M.D.
Wm. Wallis Ord, M.D., B.Ch.
Leonard Arthur Bidwell.
Arthur J. M. Bentley, M.D.
Francis R. B. Bisshopp, M.B.
Henry Percy Dean, M.B.
Louis Albert Dunn, M.S.
Daniel Mackay Forbes.
H. Pennell Hawkins, M.D., B.Ch.
Lauriston Elgie Shaw, M.D.
1890 John Rose Bradford, M.D., F.R.S.
J. Fletcher Little, M.B.
Robert Robertson, M.D.
Ernest Solly, M.B.
James Taylor, M.D.
Francis O. Buckland, B.A., M.B., C.M.

1890 G. Somerville Robinson.
Edmund W. Roughton, B.S.
Edgar Willett, M.B.
Thomas H. Rickard Crowle.
James Calvert, M.D.
H. Roxburgh Fuller, M.D.
Arthur F. Voelcker, M.D.
Neville Wood.
W. Roger Williams.
Gilbert B. M. White, M.B., B.S.
Frederick Charles Wallis, M.B., B.C.
Alfred Parkin, M.S.
George A. Hawkins-Ambler.
James Harper, M.D.
Walter Henry Brown.
John Walter Carr, M.D.
Ernest Le Cronier Lancaster, M.B., B.Ch.
Patrick Manson, C.M.G., M.D., F.R.S.
Miles Miley, M.A., M.B.
Edgar Herbert Thane, M.B.
Charles William Chapman, M.D.
Michael G. Foster, M.A., M.B.
1891 Frederic François Burghard, M.D., M.S.
Roger Neville Goodman, M.B.
Herbert Furnivall Waterhouse, M.D., C.M.
Walter William Hunt Tate, M.D.
William Lee Dickinson, M.D.
Greville MacDonald, M.D.
J. Kingston Barton
J. Dundas Grant, M.D.
W. Kington Fyffe, M.B., B.C.
Albert Carless, M.B., M.S.
W. Bolton Tomson, M.D.
Harry Littlewood, M.D.
Hector W. G. Mackenzie, M.A., M.D.
Chas. Percival White, M.B., B.C.
Arnold Caddy.
Theodore Stacey Wilson, M.D., C.M.
1892 William Hunter, M.D.
Frank Thomas Paul.
Walter Essex Wynter, M.D., B.S.
Damer Harrison.
John Alfred Masters, M.D.
Walter Giffard Nash.
John Stephenson Selwyn-Harvey, M.D.
Ewen Carthew Stabb.
1892 Edward John Lewis, M.B., B.C.
Henry Betham Robinson, M.D. M.S.
Richard James Herbert Scott.
Howard Henry Tooth, C.M.G., M.D.
1893 John Ernest Paul, M.B.
James William Bond, M.D.
Harry Campbell, M.D.
W. Soltau Fenwick, M.D.
Ernest Alfred Snape, M.D.
Lewis G. Glover, M.B., B.C.
Solomon Charles Smith, M.D.
William Bracewell Mercer, M.B., B.C.
Robert Henry Cole, M.D.
Donald Rose Paterson, M.D., C.M.
Walter Spencer Anderson Griffith, M.D.
Alfred Theodore Rake, M.B, B.S.
Francis Charles Abbott, M.B., B.S.
James William Browne, M.B.
Edwin Harding Lendon, M.B.
George Redmayne Murray, M.D., M.A.
R. J. Bliss Howard, M.D.
Henry Jones Curtis, M.D., B.S.
Henry Albert Caley, M.D.
John Henry Dauber, M.B.
Theodore Henry Ionides, M.B., B.S.
Arthur Foxwell, M.D.
Horace George Turney, M.D.
Henry Grabham Lys, M.D.
Arthur Ernest Sansom, M.D.
John Henry Bryant, M.D.
G. F. Johnston, M.D.
Willmott H. Evans, M.D., B.S.
Charles Gibbs.
Robert Stephen Charsley.
Joseph Pearce Budgett Wills, M.D.
Arnold Lawson, M.D.
1894 Herbert Morley Fletcher, M.D.
Edward Wilberforce Goodall, M.D.
Harry Gilbert Barling.
Ernst Michels, M.D.
Charles Jerome Woollett.
StClair Thomson, M.D.
George A. Sutherland, M.D.
John Murray.

1894 Richard Henry Anglin Whitelocke, M.B., C.M.
John Attlee, M.D., B.C.
Griffith Charles Wilkin.
Thomas Hugh Dickson, M.B., B.C.
C. Gordon Brodie.
Bernard Scott.
John Porter Parkinson, M.D.
Thomas Rudolph Smith, M.B., B.C.
Arthur Pearson Luff, M.D.

1895 Herbert Murray Ramsay.
Arthur G. Phear, M.D., B.C.
Ernest E. Lewis, M.D.
Karl Grube, M.D.
Gerald R. Baldwin.
James Galloway, M.D.
F. B. Willmer Phillips, M.B.
Charles Henry Hough.
Leonard George Guthrie, M.D.
James Ernest Lane.
Frank J. Wethered, M.D.

1896 Richard Ackerley.
Charles Spurrell.
Cuthbert Sidney Wallace.
Charles R. Box, M.D.
Walter Stacy Colman, M.D.
Edward Ward, M.B.
Norman Dalton, M.D.
Fred. Parkes Weber, M.D.
Francis James Steward, M.B., B.S.
William Bezly Thorne, M.D.
Douglas Drew, M.D., B.S.
Surg.-Capt. W. R. Crooke-Lawless, A.M.S., M.D.
William Henry Crosse, M.D.
William Elliot Risdon, M.D.
Robert Milne Beaton, M.B.
Priestley Leech, M.D.
Ebenezer Stanley Smith, M.D.
G. H. A. Comyns Berkeley, M.B.
Edward Cooper Bentham.
Robert Herbert Mills-Roberts.
Jonathan Hutchinson, jun.
Frederick E. Batten, M.D.
Farquhar Mac Rae, M.B.
Rutherford John Pye-Smith.
George Templeton.
Bertram Louis Abrahams, M.B., B.Sc.

1897 Raymond H. P. Crawfurd, M.D.
John Henry Deane.
Percy Furnivall.
Arthur Whitfield, M.D.
Frederick William Forbes Ross, M.D.
Harold Low, M.B.
Charles Ralph Keyser.
George Bertram Hunt, M.B.
Hugh Roger-Smith, M.D.
Alfred Herbert Tubby, M.S.
Charles Lamplough.
H. Campbell Thomson, M.D.
Alfred P. Hillier, M.D., C.M.
F. Sherman Toogood, M.D.
E. Claude Taylor, M.D.
Charles Humphrys.
Charles Sempill de Segundo, M.B.
Arthur John Whiting, M.D.
Alfred Downing Fripp, C.B., M.V.O, M.S.
Chichester Gould May, M.D.

1898 J. J. Perkins, M.D.
James Jackson Clarke, M.B.
Ernest Herbert Cobb.
Sydney Backhouse Hulke.
Thomas Herbert Kellock.
William Darley-Hartley.
Robert Jones.
Arthur Philip Beddard, M.B.
Alfred Ernest Russell, M.D., B.S.
William Cecil Bosanquet, M.D.
Frederick Ingor Willey, M.B.
Jonathan F. C. H. Macready.
Arthur A. H. Partridge, M.B., M.Ch.
Leonard Avery.
Herbert Challice Crouch.
William Turner, M.B., B.S.
Harold Batty Shaw, M.D.
Joseph Stewart Richards, M.D., B.S.
William Henry Breffit Brook, M.D., B.S.

1899 George Edward Rennie, M.D.
V. Warren Low, M.D., B.S.
Rayner Derry Batten, M.D., B.S.
Charles E. Forbes Mouat-Biggs.
Rutherford Morison.
R. Murray Leslie, M.D.
Herbert Tilley, M.D.
Charles Owen Fowler, M.D.
Cyril Ogle, M.B.

1899 Charles Herbert Fagge, M.S.
Claude Somerville Hawkes.
Henry Stephen Sandifer, M.D., B.S.
Marcus Marwood Bowlan, M.B.
Alfred Eïd, M.D.
Karl Fürth, M.D.
Wm. Gream Stone, M.B.
Andrew Duncan, M.D.
Edmond Lucien De Chazal, M.D.
Henry Roe Walker.
Gustave Schorstein, M.B.
Edward Collingwood Andrews, M.D.

1900 Berkeley George Andrew Moynihan, M.S.
Harry Cooper, M.D.
George Augustus Tullis, M.D.
Griffith Lloyd Roberts, M.B., C.M.
Edward Percy Paton, M.S.
Kenneth Scott, M.B.
George Steele Perkins, M.D.
John Arthur Hayward, M.D.
Wilfred J. Hadley, M.D.
John Fawcett, M.D.
Arthur Bousfield, M.B.
J. S. Risien Russell, M.D.
Cecil H. Leaf, M.B.
Josef Paul Zum Busch, M.D.

1901 Ernest George Leopold Goffe, M.D.
Arthur Hamilton Nicholson Lewers, M.D.
Charles Reissmann, M.B.
Adrian Caddy, M.B.
Herbert William Carson.
Thomas Henry Morse.
Laurie Hugh McGavin.
F. U. Menzies.
Thomas Rupert Hampden Bucknall, M.S.
Edward Angas Johnson, M.B.
Percival Horton-Smith, M.D.
Sir Hugh Reeve Beevor, Bart., M.D.
Theo. N. Kelynack, M.D.
Walter S. Frith, M.B.
Thomas Carwardine, M.S.
Bowie Campbell Gowan.
John J. Douglas, M.D.
Lieut.-Col. Alfred Keogh, C.B., M.D.
Harold Shuttleworth Barwell.

1902 George Beckett Batten, M.D.
Charles Henry Fennell, M.B.
H. L. Barnard.
Thomas Percy Legg, M.B.
Andrew Elliot, M.D.
Sir James Reid, Bart., G.C.V.O., K.C.B.
F. W. Price, M.B.
Alfred Ernest Sears.
Reginald F. Jowers.
Cecil Marriott, M.B.
Sidney Herbert Snell, M.D., B.S. D.P.H.
Edward Farquhar Buzzard, M.B.
Donald John Armour, M.B.
Edred Moss Corner, B.Sc., M.B.
Herbert French, M.B.

REPORT

OF THE

COUNCIL OF THE CLINICAL SOCIETY.

MAY, 1902.

THE COUNCIL has to report that the work of the Society during the session has been very successful.

The supply of papers has been good, and several valuable contributions have been made by provincial members, a fact which the Council regards with special satisfaction.

The Clinical Evenings have proved very attractive, and the method of conducting the meetings introduced last session continues to work well.

After careful consideration the Council has decided to recommend a revision of one of the Rules of the Society (Section XII, Rule 8), which prohibits the publication elsewhere of all papers read before the Society. The early publication of new facts is equally in the interest of the profession at large and of individual authors, and the Council believes that a recognition of this fact would add to the popularity of the Society. Precedents for the proposed change are to be found in the action of other societies, viz. the Royal Medical and Chirurgical, the Medical, and the Pathological Societies.

The Council accordingly proposes that authors of papers read before the Clinical Society be permitted to publish them afterwards in the medical journals, due acknowledgment being made of the fact that the papers have previously been read at the Society.

In accordance with the requirements now added to the Rules of the Society (Section IX, Rule 1) that the Treasurer "shall from time to time hand to the Trustees all composition fees for investment," the sum of £40 Consols has been purchased with the proceeds of such fees received since the last Annual Meeting, thus increasing the Society's invested funds to £640.

The number of Ordinary Members is 602, of Honorary Members 15, giving a total membership of 617. During the session 22 Ordinary Members have been elected.

The Society has lost eleven members through resignation, and death has removed one Foreign Honorary Member, Professor von Ziemssen, and four Ordinary Members, viz. Mr. Holthouse, Sir William MacCormac, Bart., Dr. Henry Sutherland, and Dr. James Mann Williamson.

The Council recommends the following alteration in the Rules of the Society:

Rule 8, Section XII, is deleted, and the following substituted:

"Authors of papers read before the Clinical Society are permitted to publish them afterwards in the medical journals, due acknowledgment being made of the fact that the papers have previously been read at the Society."

THE CLINICAL SOCIETY OF LONDON.

Statement of Cash Receipts and Payments from the 1st May, 1901, to the 30th April, 1902.

FREDERICK TAYLOR, Esq., M.D., *Treasurer.*

RECEIPTS.	£	s.	d.
Balance, 1st May, 1901	193	15	1
168 Subscriptions received by Collector £176 8 0			
192 „ „ Bank 201 12 0			
360	378	0	0
5 Subscriptions at 10s. 6d.	2	12	6
19 Admission Fees at £2 2s.	39	18	0
4 „ „ at £1 1s.	4	4	0
5 Composition Fees	26	5	0
1 Composition Fee (*Transactions*)	10	10	0
Sale of *Transactions*	22	7	2
Dividend on Consols (£600)	15	11	0
	£693	2	9

PAYMENTS.				£	s.	d.
Cost of *Transactions*, Vol. XXXIV:						
Printing, paper, binding, delivery, &c.	£145	11	8			
Illustrations	76	1	3			
				221	12	11
Meetings:						
Rent of Rooms	105	0	0			
A. Tapson (attendance)	7	10	0			
Refreshments	33	8	0			
				145	18	0
Cost of Purchase of Consols (£40)				37	19	6
Treasurer and Hon. Secretaries:						
Secretarial assistance	26	5	0			
Commission to Collector	15	15	0			
				42	0	0
Stationery and Printing	80	16	11			
Reprint of Dr. Tooth's Paper on Enteric Fever	3	1	0			
				83	17	11
Petty Expenditure				10	8	9
Balances:						
At Bank	147	5	5			
Petty Cash	4	0	3			
				151	5	8
				£693	2	9

HOWARD MARSH, *President.*
FREDERICK TAYLOR, *Treasurer.*
PERCY KIDD, *Hon. Secretary.*

Examined with the vouchers and found correct,
EDGAR WILLETT.
ARCHIBALD E. GARROD. } *Auditors.*

COMMUNICATIONS.

I.—*A note on a Painful Condition of the Twelfth Pair of Ribs.* By J. Jackson Clarke, M.B.Lond. *Read October* 11, 1901.

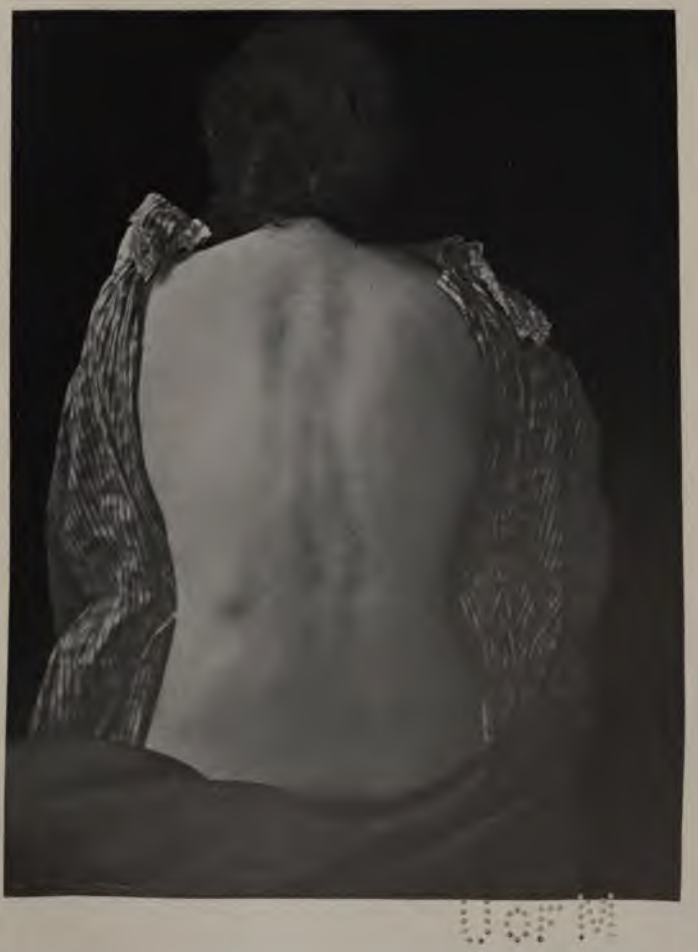
L. S., a nursemaid, æt. 17, came under my care at the North-West London Hospital in November, 1899. She complained of pain in her back and consequent inability to do her work. The patient's face was puffy, and her skin pale and yellow. She complained of constipation. On examination I found that the antero-posterior curves of the spine were exaggerated. The spinous processes were tender to pressure, and the movements of extension and rotation of the spine caused pain. The tips of the twelfth ribs were prominent, and pressure upon them caused pain. The deformity of the spine was fixed, *i. e.* it did not disappear when the patient lay down. In order to correct the spinal deformity and to enable the patient to resume her work without risk I decided that, in addition to general treatment and systematised exercises, it was advisable for her to wear an efficient antero-posterior spinal splint. This I applied in December, 1899. The pain in the spine was thus at once relieved, and the twelfth ribs were at the same time shielded by the instrument from pressure. The spinal deformity gradually diminished, so that in November, 1900, the patient, who had continued at work during the treatment, was able to dispense with the instrument, keeping it by her, however, in case the pain should recur and render the temporary use of the instrument advisable. The twelfth ribs still remained prominent,

and on leaving off the instrument the patient again experienced a considerable amount of pain in them. I had the patient's corsets cut away over the prominent ends of the ribs, in the hope that this would afford relief. The pain persisted in spite of this measure, and it became clear that operative treatment was most likely to remove the pain, which I had no doubt was due to the pressure exerted by the patient's dress upon the prominent extremities of the ribs, imparting movements at the joints between the ribs and the vertebræ, these joints being the seat of osteo-arthritic changes. A photograph of the patient's back taken on the day of her admission to hospital (January 9, 1901) shows the prominence caused by the left twelfth rib. That on the right side was equally prominent, but it does not show, owing to the right side being in shade. On January 24 I cut down on each rib and removed the part that projected beyond the erector spinæ muscle—one inch in each case including the cartilage, the periosteum, as well as bone and cartilage, being taken away. The deep parts I stitched together with fine silk and closed the skin incisions with silkworm gut. The patient had no *post*-operative trouble except on the left side, where she complained of pain along the course of the twelfth dorsal nerve. This troubled her chiefly when she laughed. The symptom had disappeared when she left the hospital on February 25. She resumed exercises three weeks after the operation, and when discharged she was well in every respect.

The only abnormal feature that I observed in the portions of ribs that were removed was that the terminal cartilage was sharply bent forward. The seat of the pain was not, however, at the tips of the ribs, but deeper, at the costo-vertebral joints.

DESCRIPTION OF PLATE I.

To illustrate Mr. Jackson Clarke's paper on A Note on a Painful Condition of the Twelfth Pair of Ribs.



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II.—*Case of Displaced Strangulated Femoral Hernia in which the sac with its contents was displaced downwards and inwards, through an opening at the lower end of the femoral sheath, in front of the adductor muscles, on the inner side of Scarpa's triangle.* By THOMAS BRYANT. *Read October* 11, 1901.

ON April 3, 1901, Dr. Robinson, of Wandsworth Common, brought a patient into Bolingbroke Hospital whom he asked me to see. She was a married woman, thirty-two years of age, who had been troubled with a femoral rupture for about five years, and had not worn a truss. The rupture had come down every now and then, and she said she had never experienced difficulty in its reduction, although on questioning her husband later on, he was sure that she had often used much force in reducing the rupture, and particularly on March 29, two days before her present illness and the attendance of Dr. Robinson.

Her illness was supposed to date from March 31, or three days before her admission, and it commenced as a "bilious attack," to which she was rather prone, by sickness and headache, and it was not till three days had passed, and the vomited matter had become stercoraceous, that the possibility of the case being due to her reduced hernia had been realised and my advice was sought.

When I saw her on April 3 she was in an extreme state of collapse, and it was clear that life could not be saved. Her abdomen was distended, but not tense, and there was no swelling in the right femoral region, which was given as the seat of her hernia, although pressure with the pulp of the index finger over the right femoral canal elicited pain. No enlargement of the thigh was noticed in the region into which the hernia was subsequently traced.

An exploratory operation in the femoral region was, however, determined upon, based upon the good old practice of first examining, in every case of intestinal obstruction associated with a hernia, the region in which the hernia existed.

The operation was carried out in the usual way by a vertical incision of the soft parts over the crural canal, and

the exposure of the crural sheath, which did not seem to be expanded, although on palpation over the femoral canal it was thought to be slightly distended. The sheath was then opened with great care, when a small piece of tissue, which looked like omentum, was seen, but no bowel; the femoral ring was quite clear, and upon a director was slightly enlarged upwards and inwards; as a result of this action a piece of pale intestine which before had been collapsed, and had not been seen, suddenly became distended and arched forwards in the femoral canal, its distal end passing downwards through a narrow opening at the apex of the femoral sheath, an inch or an inch and a half below the femoral ring. At this point the bowel was strongly held and strangulated; upon a grooved probe director this seat of stricture was divided, when I was enabled to draw out of a space situated below and to the inner side of the opening in the femoral sheath at least four or five inches of highly congested but not otherwise injured intestine, and to reduce it easily into the abdominal cavity.

The operation was thus completed, but the patient sank as expected, and died a few hours later.

A partial *post-mortem* examination could alone be obtained, but from the specimen which was secured by Mr. Lyster, the house surgeon of the hospital, and which, as dissected and mounted by Mr. Shattock, is now in the Museum of the Royal College of Surgeons of England, Preparation 2677A, and from the drawing accompanying this paper, it seems clear that the sac of the femoral hernia had been forced through a rupture of the femoral sheath at its lowest part or apex into the connective tissue of the thigh on the inner side of Scarpa's triangle, and that the seat of strangulation was at the neck of the sac, which was not, as is usually the case, at the femoral ring, but at the seat of rupture of the femoral sheath through which the hernial sac had been forced.

Remarks.—The case which has just been read is clearly a very unusual one, and, as such, is worthy of publication; indeed, I have never seen or read of a femoral hernia being displaced as this must have been into the fatty connective tissue of the inner side of the thigh, with the seat of strangulation being at least an inch and a half below the femoral ring, its usual seat, and situated at the mouth of the opening in the femoral canal through which it was probably forced.

Had there not been a distinct history of an old femoral hernia in this case, there would not have been any local

indication to lead the surgeon to suspect that the intestinal obstruction which the symptoms suggested was due to such a cause, for there was a total absence of all swelling, and the sign of pain on pressure over the femoral canal stood alone.

Under these circumstances an abdominal operation might have been undertaken, and if so, it would have been useless, for the seat of strangulation was outside the abdominal cavity, and could not by such a measure have been relieved, for it must be emphasised that during the operation when the femoral sheath was first opened, the bowel which was in it was not seen, for it rested in a bloodless and collapsed condition upon its posterior wall, and it was not till the crural ring was enlarged that it made itself manifest by bulging forwards and arching downwards from the femoral ring to the seat of its stricture and the apex of the femoral canal.

This portion of exposed intestine was quite healthy-looking and pale, and contrasted strongly with the deeply congested bowel which was subsequently withdrawn from the displaced hernial sac.

The cause of the displacement of the femoral hernia in the case recorded must doubtless have been mechanical, and was probably due to the forcible attempt by the patient at reduction of the hernial tumour after its descent, the force employed having brought about primarily a rupture of the femoral sheath at its lower end, and secondarily the gradual herniation of the femoral sac with its contents into the space in the connective tissue as demonstrated by the dissected specimen. It is probable also that what the patient described as her bilious attacks were due to a partially obstructed hernia.

We have, therefore, in this case an example of a third variety of displaced femoral hernia, the *first* being the well-recognised form in which the small strangulated femoral hernia with its sac may be pressed out of sight and reduced, still strangulated behind the abdominal parietes—the one of "reduction *en masse*;" the *second* being where a large femoral hernia, by some rupture of its sac, may show itself as a tumour in the subcutaneous connective tissue above or below Poupart's ligament; and the *third* as a displaced hernia following the course of the case just described.

In the many varieties of "displaced inguinal" hernia which have been described, and particularly by Mr. J. Birkett and the reporter of this case, in his *Practice of*

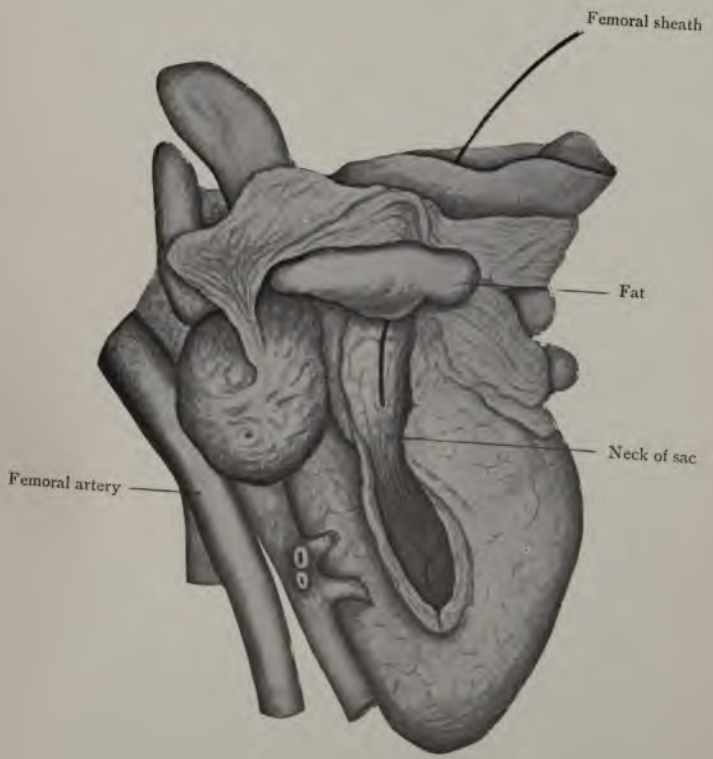
Surgery, the injurious effects of misapplied force have been fully recognised; indeed, it seems probable that in all the varieties of displaced inguinal hernia it should be regarded as their probable cause.

In the case now under consideration of femoral hernia, as in its other varieties, the same conclusion has to be drawn, and the truth of the following aphorism enforced:

"That, as in the treatment of every variety of obstructed or strangulated hernia, forcible taxis is the direct cause of all cases of displacements of the inguinal or femoral varieties, as well as of rupture of the sac or sac's contents, so forcible taxis ought strictly to be avoided."

DESCRIPTION OF PLATE II.

To illustrate Mr. Bryant's case of Displaced Strangulated Femoral Hernia.



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III.—*The Treatment of Wounded Joints.* By CUTHBERT S. WALLACE. *Read October* 11, 1901.

WOUNDS of joints, as far as treatment is concerned, seem to fall into three groups.

First.—Gaping wounds, often complicated by fracture of bone, such as are met with in railway and machinery accidents or run-over cases.

Second.—Wounds or probable wounds of the joint cavity without complications.

Third.—Doubtful wounds of the synovial cavity.

The treatment of the first class case must depend on the nature of the complicating injury, and, as a rule, the question of amputation has first to be considered. If there is extensive comminution of bone, great laceration of soft parts, and rupture of vessels, ablation of the limb is the only course. Where the injury is less than in the preceding case the surgeon must still be guided by the local condition and the likelihood of a useful limb resulting, remembering that a limb of flesh and blood, even though short and somewhat deformed, is better than a wooden one. The age of the patient has a distinct influence on the course to be taken, for a young man has a greater recuperative power than an old one, to whom the mere confinement to bed may be a serious consideration, and it is obvious that it is worth while for a young man to run a risk in order to obtain a useful limb to which it would not be worth while subjecting an old man whose time is nearly run.

Wounds or Probable Wounds of the Joint Cavity without other Complications.

Of this class there were three instances admitted to St. Thomas's Hospital during the year 1899. One died of sepsis, one recovered with a stiff knee, and one with a good joint.

CASE 1.—T. J., male æt. 61, was admitted on November 13, 1900, with a history that a wheel of a dray had passed over his left knee. On the inner side of the joint was a wound the size of the palm of the hand, and the tissues were begrimed with mud and dirt; the joint was found to have

been opened over the internal semilunar cartilage for the space of one inch. The wound was carefully cleaned, but the joint was not opened up. On the following day the joint was distended and swollen, and somewhat painful; the general condition was good. An incision into the joint evacuated sero-pus, a cultivation from which was subsequently reported by Mr. Seligmann to contain a staphylococcus and a thin bacillus. During the next twenty-four hours the patient seemed to be doing well, but at the end of that time showed signs of septic absorption, in consequence of which the leg was ablated through the lower third of the thigh, but too late to save the patient's life.

I think that considering the age of the patient amputation should have been performed directly the joint became infected, that is on the day following the accident.

Case 2.—L. S., female æt. 4, fell and cut her knee on a broken washing basin; the resulting wound was two inches in length. The wound was cleaned, but no actual opening into the joint was found. Four days later (August 3) the temperature rose to 103°. The joint was incised, washed out with saline, and closed. On August 8, as the temperature remained high, the irrigation was repeated and the wounds left open. For the next three weeks the joint was daily irrigated, until at the end of this time the temperature became normal. Dense fibrous ankylosis resulted. A tube of serum jelly, inoculated on August 3, remained sterile.

September 10, 1901.—Complete fibrous ankylosis of the joint is present.

Case 3.—A. W., male æt. 12, fell down on the pavement and sustained a wound to his knee-joint. He did not apply at the hospital until three days later (July 27, 1899), when the joint was found distended. The temperature was 101° F. During the process of cleaning the wound by cutting away all the dirty tissue it was found that the wound had opened the joint. The cavity was therefore irrigated with saline fluid and sutured.

July 29.—Temperature remained high, and the joint was again opened and washed out and a drain left in for one day.

August 1.—Joint again irrigated under an anæsthetic, and incisions made into the cellulitis about the joint. For the next three weeks the temperature fluctuated between 104° F. and normal.

September 6.—Flexion to 45° was possible, and the wounds practically healed. (Culture taken from the joint, July 29, 1899, was sterile.)

September 10, 1901.—The patient has a good moveable knee, with practically no limitation.

When one is called upon to treat a case of a recent wound over a joint without precise knowledge that the joint is actually opened, three points appear worthy of consideration:

1. The actual method of cleaning the wound.
2. Whether it is worth while searching for the actual opening into the joint.
3. If the opening into the joint is found, whether it is better to irrigate the joint or await developments.

1. *Method of cleaning the wound.*—This, I believe, is best done by a careful process of cutting away, as far as possible, all soiled tissue, and the use of sterile saline solution simply for its mechanical effect.

Any rubbing of the main surface is to be deprecated, since it is extremely likely to rub into the tissues as much dirt as it removes.

2. *Question of searching for the actual wound of the joint.*—This should not be carried too far, because even if the wound is found no great advantage can accrue, and much harm can be done if a fresh opening is made.

3. *Should the joint be irrigated if found open?* The answer to this must in most cases be in the negative, because of the danger of carrying in organisms from the dirty external wound, and also because it is extremely unlikely that much good can be done even if antiseptics are used (indeed, they may themselves be a source of irritation to the joint). On the other hand, if the joint is found to contain dirt, or if the synovial membrane or cartilage have dirt ground into it, then the joint should be sufficiently opened to allow of the removal of the dirt and soiled tissue.

Speaking generally, I think an attempt should be made in these cases to shut off the joint cavity from the open wound, and to this end the joint capsule should be sutured up.

Doubtful Wounds of the Synovial Cavity.

The third class of case, namely, that of doubtful wounds of joints, is perhaps the most interesting of all, and in this class must be included penetrating or perforating wounds,

because of the difficulty of really ascertaining whether the joint cavity has been actually entered.

I have notes of five examples of this injury lately; they were all treated in the same manner, and show, I think, satisfactory results.

Few surgeons would at the present time advocate the opening up of the wound to make a definite diagnosis, and the interest turns on the treatment to be adopted when the joint appears to have become infected.

Wm. R., male æt. 16, was wounded on the inner side of the patella by a steel pitchfork on June 25, 1899. The joint became swollen and then subsided. On June 30 the joint again became swollen, and on July 3 the patient was admitted to the hospital. The temperature was 101° F. The joint was opened, sero-pus evacuated, and after saline irrigation the capsule was closed with sutures. The temperature at once fell, and on July 19 the patient was discharged with a normally moveable joint, except for a little limitation of flexion. A broth tube inoculated from the contents of the joint remained sterile.

September 10, 1901.—Patient has a good moveable joint.

H. P., male æt. 11, was admitted on August 3, 1899, with a punctured wound of his knee-joint. On the same day the joint was incised, irrigated with saline fluid, and then closed with sutures, except for a small gauze drain, which was removed on the next day. On August 11 the patient was discharged with a good moveable joint.

September 10.—Boy is now in the Royal Navy, and according to his father's account the joint is normal.

A. V., male æt. 6, fell on to a meat hook on June 9, and sustained a small punctured wound over the knee-joint. On June 11 the knee was stiff and a little swollen, but the other symptoms were not severe enough to make it appear probable that the joint was affected. He was accordingly treated with lead lotion and a back splint. The following day the joint was greatly swollen and extremely painful. Temperature 100° to 102°. The joint was therefore opened and sero-pus evacuated. The joint was then irrigated with saline fluid and closed, except for a small gauze drain that was left in for twenty-four hours. On June 23 the patient was discharged with the wound healed. The movement in the joint was normal. No culture was taken.

Subsequent history is unknown.

For permission to use this case I am indebted to Mr. H. B. Robinson.

B. H., æt. 11, was admitted into St. Thomas's Hospital on August 31, 1899. The day previously he had fallen down and run a rusty iron spike into his knee. The wound was a small one, and no opening into the joint was found. The wound was carefully cleaned by cutting away all soiled tissue and closed with sutures. On September 6 the joint became tensely swollen and very painful. The temperature was only 100° F., but the pulse was rapid, and from the general condition of the boy it seemed probable that a septic arthritis was present. The joint was opened and turbid serum evacuated. The cavity of the knee was irrigated with saline fluid and the joint closed completely. An uninterrupted recovery followed, and the patient was discharged with a moveable joint on October 4. A cultivation was taken from the fluid evacuated from the joint, and the resulting growth was reported by Mr. Singer to be one of the *Staphylococcus pyogenes albus.*

The after-history of this case cannot be traced.

Wm. B., male æt. 37, while breaking a piece of wood across his knee ran a rusty nail into the joint. The following day, November 5, 1899, the patient was admitted with a painful and swollen knee-joint. The temperature was 101°. There was some doubt at the time as to whether the swelling was due to a simple or septic arthritis, and the joint was tapped and a culture tube (broth) inoculated. This, unfortunately, was broken. The temperature gradually dropped to 100° F., but on November 8 it was decided to open the joint, and this was accordingly done. After lavage with 1 in 1000 perchloride of mercury the wound was closed, but a drainage-tube was left in the joint for twenty-four hours. On the following day the temperature rose to 101°. There was a considerable amount of discharge, and the joint was daily irrigated with saline fluid, until, on December 7, the wounds were closed, but considerable tumefaction of the joint tissues remained, and a little latent mobility could be obtained. On January 17 passive movement was performed under an anæsthetic.

February 2, 1900.—Knee could be flexed to a right angle. Discharged February 2.

September 10, 1901, there was free movement for an angle of 35°, but at this point the leg is suddenly and definitely arrested.

I am indebted to Mr. Pitts for this case, under whose care the patient was admitted.

In four of the above five cases one irrigation of the joint was sufficient, in the fifth the infection was more virulent, and the joint was for some days washed with saline fluid. This was the only case of the series in which an antiseptic was used.

The good results in these cases are rather surprising, and it might be considered that the effusion into the joint was a simple traumatic synovitis. Against this it must be remembered that the joints were not opened until sufficient time had elapsed to form a deliberate opinion.

In two of the cases cultivations of the joint fluid were made: one remained sterile, and in one a growth of staphylococcus was found; both, however, did equally well.

The joints were practically closed in all cases, as when drainage was employed it was only for twenty-four hours.

Mr. F. C. Wallis, in a paper entitled "Perforating Wounds of the Knee-joint," which he read before this Society in 1898, expressed an opinion that in septic arthritis it is better to avoid the use of drainage, at any rate for an extended period. These cases, I think, bear out the contention there expressed.

It is often a matter of difficulty to make a differential diagnosis between septic and simple arthritis when a wound has been received over a joint, and should it be determined to open the joint, lavage and suture is certainly a less severe procedure than drainage, and much less damage is done if the case was really one of simple traumatic synovitis.

I would suggest that the best treatment in septic arthritis is lavage and suture of the joint in the first instance, or this may be repeated a second time if the symptoms do not show signs of abatement. If, after this, the temperature remains high and the joint swollen and painful, the incisions may be reopened and daily lavage of the joint practised with or without an anæsthetic. It is really a very moot point whether any amount of irrigation, no matter of what the irrigating fluid may consist, will really be of any value when once the synovial membrane is infected and swollen, since the disease has by that time ceased to be intra-articular and has become peri-articular.

Multiple incisions with many drainage-tubes have, in my experience, nothing to commend them, and even more deleterious is continuous irrigation through drainage-tubes introduced into the joint cavity. Granulations almost immediately spring up round such tubes, which then do little except to drain the tract they have made for themselves, and may even cause ankylosis in a joint that without them would have recovered with some movement.

I think it must be recognised that when early lavage has failed to arrest the inflammation, and the infection has reached the tissues outside the synovial membrane, surgery can do but little to aid nature in its struggle with the infecting germs, except in giving exit to abscesses which may form about the joint, and possibly in ablation of the limb.

In the first three cases cultures were made from all the joints. In the fatal case a staphylococcus was formed, and in the second case (L. S.) the serum jelly remained sterile, but this case ended in complete fibrous ankylosis, possibly due to the frequent disturbance of the joint by irrigation. In the third case the culture remained sterile and a moveable joint resulted, although lavage was employed three times.

In the second series two cultivations were made from the joints: one remained sterile, and in one a growth of staphylococcus was found; in both, however, a moveable joint resulted.

Conclusions.

1. That it is not expedient to search too vigorously for the wound into the joint.
2. That if the joint be found open it is better to close it without irrigation, and await developments.
3. That if the joint become infected it should be incised, irrigated, and closed, and that this process may be repeated.
4. That if the infection continue and synovial membrane and joint capsule become swollen, little good is likely to result from the employment of multiple drainage-tubes or continuous irrigation.
5. That the process of cleaning either a wound or a joint must be mechanical, and that therefore soiled tissues should be carefully removed with knife and scissors, and that it is useless to employ irritating chemical solutions.

IV.—*The Treatment of Ascites by Fixation of the Omentum and the formation of Adhesions.* By C. Mansell Moullin. *Read October* 11, 1901.

THE cases which I wish to bring before the Society this evening do not need any lengthy description. They are, all of them, ordinary cases of ascites dependent upon cirrhosis of the liver, such as occur only too frequently. The point which I wish to raise is the advisability or not of treating such cases by what is known as the Drummond-Morison method of operating—suturing, that is to say, the omentum to the anterior abdominal wall, and creating adhesions between the surface of the liver and the parietal peritoneum, in the hope of relieving the portal circulation by establishing an alternative route, and perhaps of enabling the superficial layers of liver-cells to perform their duties better by diminishing the congestion from which they suffer, and improving the circulation around them.

My cases are five in number. They were all admitted into hospital under the care of my colleagues, Dr. Sansom and Dr. F. J. Smith, and subsequently transferred to my wards for operation. The first was that of a woman æt. 53, who came into hospital on May 15, 1899. Her illness had commenced five months before with swelling of the legs. The abdomen had begun to enlarge about three weeks before, and for that time she had been confined to bed. The patient's weight was 15 st. 3 lbs. The breath was very short, and the lungs were emphysematous. The bowels had been open rather too freely. The appetite was bad. The abdomen was distended and the abdominal wall œdematous. The liver was apparently enlarged, smooth, and hard. The urine was small in amount, acid, 1022, and contained a trace of albumen. There was abundant history of alcoholism, but none of syphilis. She was tapped on the medical side without relief, and on June 6 I performed median laparotomy, let out an enormous quantity of ascitic fluid, sutured the omentum to the anterior wall, and gently rubbed the surfaces of the liver and the parietal peritoneum. The patient was apparently none the worse for the operation, but fluid began

to collect again very rapidly. On June 11, 230 ounces were let out, and on the 17th 25 ounces more. After this the abdomen remained lax; but on the 22nd the breathing became very embarrassed, and on the 26th 55 ounces of blood-stained fluid were withdrawn from the left pleural cavity. This relieved the dyspnœa for a time, but the fluid collected again in the pleura; the temperature rose, and although temporary relief was obtained again by aspirating the chest, the patient died on July 7. At the autopsy the coronary arteries were found to be very atheromatous. There was a large amount of fluid in the left pleura, and a moderate amount in the abdomen. The intestines were matted together and to the abdominal wall by recent adhesions. The liver weighed 3 lbs. 4 oz., and showed a very advanced stage of atrophic cirrhosis. The kidneys and spleen were healthy.

The second case, that of a man æt. 46, also terminated fatally. His illness had only lasted ten weeks. He was slightly jaundiced and his arteries were rather atheromatous. The abdomen was very much distended. The liver and spleen could not be felt, but the liver dulness was much diminished. The bases of both lungs were congested, and the urine was thick and dark, bile-stained, with a specific gravity of 1034, but with no sugar or albumen. After preliminary tapping, the same operation was performed as in the last case, but without any benefit. There was no shock or peritonitis. A certain amount of fluid re-collected, but not sufficient to require treatment. The patient simply became weaker and weaker, and died from exhaustion at the end of a week. At the *post-mortem* examination the liver was found in an advanced stage of cirrhosis, weighing 4 lbs. 2 oz., and there were numerous recent adhesions between its capsule and the abdominal parietes.

My third case was that of a man æt. 40, who was operated upon in the autumn of 1899. His illness was of only six weeks' duration, and began, according to his own account, with diarrhœa and distension of the abdomen. He was very short of breath, but had been able to keep on with his work. The urine was acid, 1025 specific gravity, and did not contain any albumen or sugar. The heart and lungs were healthy. The abdomen was very much distended with fluid, and the legs were slightly œdematous. A similar operation was performed, about two gallons of ascitic fluid being let out, and the wound closed. The fluid soon collected again, and was let out by means of a Southey's trocar on several

occasions. Finally the patient left the hospital relieved, and my house surgeon, who saw him last week, reports that after lying up for some time he has resumed his work, that there is no return of the fluid, that he has had one attack of hæmatemesis last winter, and that he feels absolutely well at present, though his left leg swells a little at times.

The fourth case was that of a woman æt. 38, with practically the same history. Abdominal distension, four months' duration, feet swollen, urine acid, 1022, no albumen or sugar, but the quantity small; pulse 120, respiration 32, but no evidence of any thoracic complications. The same operation was performed in November last year. Seventeen days afterward 120 ounces of fluid were withdrawn by means of a Southey's trocar, and shortly afterwards she left the hospital to attend as an out-patient, but I have not been able to trace her.

The fifth was also a woman æt. 38, but the history of the illness was a much longer one. The abdomen had gradually been getting larger for the last four years. The appetite was good and the bowels regular, but for the last three years she had been steadily losing flesh. The urine was acid, 1040, and did not contain albumen or sugar. The quantity passed was about 25 ounces per day. She had been tapped twice previously, and a large quantity of fluid evacuated. An operation similar to that already described was performed in September, 1899. The wound healed readily, but the fluid re-collected, and was let out on several occasions while patient was in hospital. She was discharged in October, 1899, and I saw her about a week ago. The fluid had continued to collect, and tapping had been repeatedly performed until the end of December, 1899. Since then she had not been tapped, and she described herself as perfectly well, as well as she ever had been in her life.

Of these five cases, therefore, two died, one left the hospital relieved, but has not been traced, and the others are, according to their own account, perfectly well two years after the operation.

In discussing the merits of this operation I do not think much stress should be laid upon the unfavourable result of the first two cases—those which terminated fatally. They were plainly unsuitable. In both the disease had advanced too far, and in both other organs were involved as well. The good effect of this operation cannot possibly show itself at once. It requires time for adhesions to form, and for the

vessels in those adhesions to enlarge sufficiently to make an appreciable difference to the portal circulation and in the nutrition of the hepatic cells. It is clear, therefore, that if the operation is to be of any benefit, it must be confined to patients who are still in the early stage of the disease, in whom the liver has not as yet undergone a hopeless degree of degeneration, and in whom the other organs essential to life have not yet become seriously affected. Those two patients, whose cases I have included because I do not think it fair to leave any out, unhappily had passed this stage. It was too late. But the failure of the operation in these two cannot be used as an argument against its employment in other circumstances, where the conditions are more favourable; and from what happened in my other cases, and from those which have already been published, I am convinced that, provided a suitable and judicious selection is made, this operation may prove of great service; of course it does not cure cirrhosis, or pretend to cure it, but it can relieve one of its most distressing consequences, a consequence that aggravates the course and the rapidity of the disease which causes it, and often renders the balance of life that is left scarcely worth living; only the operation must be performed sufficiently early.

The question really turns upon the view that is taken of the causation of the ascites. If the ascites is of mechanical origin there is no doubt that the operation is capable of giving relief. There is the clinical evidence, familiar to every one, that a certain proportion of patients suffering from cirrhosis who are treated by repeated tapping get well, even after the number of tappings has reached twenty or thirty, or even more; and there is the pathological evidence of the enlargement of the ordinary anastomotic channels between the radicles of the portal and systemic circulations, and of the very great development of accessory channels in the freshly formed adhesions,—as, for instance, was shown most plainly by one of Morison's cases which died from an operation for ventral hernia two years after the omentum had been fixed. If, on the other hand, the ascites is held to be not mechanical, or not wholly mechanical, in origin, but to be due to the impairment in activity of the liver-cells caused by defective blood-supply, it is equally the fact that the only hope of restoring the activity of those cells, and giving them a better blood-supply, consists in establishing a number of vascular adhesions between the contiguous surfaces of the

liver and the parietal peritoneum at a period when compensatory growth is still possible.

The operation is not one that is attended by any serious degree of risk. It is true that the mortality, as judged by statistics, has been very high up to the present time, but this is always the case with new operations, and is due to the fact that many of the cases were not suitable, either because there was no cirrhosis or because other organs were gravely involved, and that the operation has been performed as a last resource, when everything else had been exhausted, and the patient was practically dying. With better selection and earlier operation there is no reason why the mortality should be appreciably higher at all than that of exploratory laparotomy. So far as the technique is concerned, there is very little to say. I have always employed a median incision above the umbilicus, and I cannot see how any other can surpass it, either in safety or convenience. The whole surface of the liver can be easily reached through it and rubbed with a gauze pad, which is quite sufficient to cause the formation of adhesions, and the omentum can be drawn up and fixed transversely across the abdomen by sutures inserted from the parietal surface of the peritoneum. A firm binder is fastened afterwards around the upper part of the abdomen to keep the walls as quiet as possible, and the patient is encouraged to sit up as soon as he can. I see no object in employing drainage if the abdomen is fairly well emptied, while there is always a certain amount of risk that the wound will become septic, as actually happened in one recorded case. It is true that the fluid collects again, but it collects in the lowest part of the abdomen, and does not disturb the formation of adhesions between the omentum and liver on one side and the parietes on the other. If at any time it becomes excessive it may be drawn off, and, as shown by the history of those cases which recover, this may require to be done repeatedly in the first two or three months, until, in short, the anastomotic channels have grown wide enough.

Ascites is a late and, as a rule, a very serious symptom in cirrhosis. It means that the power of resistance of the patient has begun to give way. If, therefore, the cirrhosis is not complicated by other disorders, and if the accumulated fluid is not absorbed quickly and completely under the influence of such drugs as iodide of potash, it seems to me more rational to try and establish an efficient collateral cir-

culation without further delay by an operation which, under such conditions, is not attended by any grave degree of danger, than to allow the patient to drift on in a state of perpetually increasing misery, relieved now and then by tapping, until, in the vast majority of cases, his strength sinks so low that it is too late to do anything more than watch the progress of the disease.

V.—*The Operative Treatment of Cancer of the Pyloric Portion of the Stomach.* By B. G. A. Moynihan, M.S. *Read November* 8, 1901.

THERE is a general agreement among surgeons that the operative treatment of malignant disease of the stomach is very unsatisfactory. The immediate mortality resulting from pylorectomy or partial gastrectomy is certainly a diminishing one, but in a strikingly large proportion of cases the health of the patient is restored for only a brief period; recurrence of the disease is soon observed and rapidly proves fatal.

Hemmeter, in a recent article upon "The Early Diagnosis of Cancer of the Stomach," says, "No matter what our methods of treatment, regardless of at how early a date or with what skill it is executed, the cases treated will eventually and inevitably suffer from a recurrence of the disease. This is an actual fact in 99 per cent. of all gastric cancers that have undergone operation."

The examination of the records of cases operated upon proves decisively that the disease, in the majority of instances, returns locally—returns, that is, either in the stomach-wall or in the adjacent glands. Lindner records twenty-eight recurrences after removal; of these fifteen were local, twelve metastatic, and one glandular. In order, therefore, to prevent local recurrence it is necessary to remove a larger portion of the stomach, and to ensure that the glands most likely to be affected are, at the same time, taken away.

The classical description of the operation of pylorectomy, which has been handed down, with trivial alteration, from Billroth's time, is probably followed in most of the operations of the present day. If the anatomy of the lymphatic vessels and glands be studied, and if the mode of invasion of the stomach by the primary growth be appreciated, it will be found that the operations of Billroth leave unfulfilled certain essential features for success.

Mode of invasion of the stomach by the primary growth.—The researches of Borrmann and Cunéo have shown that the most common point of commencement of carcinoma in the

stomach is 2 to 4 cm. from the pylorus, on or near the lesser curvature. From this point the disease spreads gradually, but more rapidly and to greater extent towards the body of the stomach than towards the duodenum. On the stomach side the growth is not equal in all directions. There is a pronounced tendency for the induration to spread towards the curvatures. As a rule, both curvatures are affected almost equally. In an examination of sixty-three specimens Borrmann obtained the following results:

In thirty-two cases both curvatures were equally affected.

In seven cases the lesser curvature was more extensively affected.

In nineteen cases the greater curvature was more extensively affected.

In three cases only the lesser curvature was affected.

In one case only the greater curvature was affected.

In one case both curvatures were free, the body only being affected.

Lebert gives the following table to show the relative frequency of affection of the various regions of the stomach:

Pylorus	54 per cent.
Lesser curvature . . .	16 „
Cardia	9 „
Anterior wall . . .	3 „
Posterior wall . . .	4 „
Both walls . . .	4 „
Greater curvature . .	4 „
Diffuse	6 „

Towards the duodenum the spreading is slower and invariably less extensive. From Rokitansky to Cunéo all investigators have emphasised the fact of the integrity of the duodenum. Invasion of the duodenum, however, does occur to a slight extent, as shown by Carle and Fantino, and must be reckoned with when an ideal operation is planned. If sections are made at the edge of a malignant neoplasm and the slides examined, the following condition will be found:—The edge of induration corresponds to the limit of growth in the mucosa. In the submucosa the growth extends much further in a solid unbroken mass; beyond the edge of this mass isolated nodular deposits of growth extend, becoming smaller as their distance from the tumour increases. The subserous and the serous coats are implicated approximately to the same extent as the mucosa. In removing any

growth the section of the stomach must, it necessarily follows, be made very wide of the indurated edge.

Lymphatics of the stomach.—The lymphatic glands of the stomach are situated chiefly along the vessels.

1. *Coronary group.*—This chain is placed along the lesser curvature of the stomach in close approximation to the coronary artery. The number of the glands is very variable. They are continued upwards along the coronary artery as far as its origin from the cœliac axis, and there become continuous with the glands along the upper border of the pancreas. One or two small glands lie over the termination of the œsophagus.

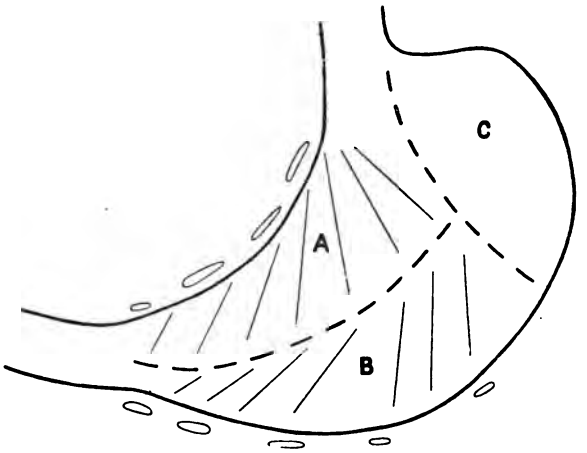

FIG. 1.—Showing the lymphatic areas and glands of the stomach. *C* is the "isolated area."

2. *Hepatic group* is placed along the hepatic artery, and reaches up to the liver; some of its members lie along the pyloric artery.

3. *Glands of the greater curvature* lie along the right gastro-epiploic artery. At the pylorus they are numerous and close together, and pass behind the stomach to the head of the pancreas, and upwards towards the hepatic group. The lymphatics of the left gastro-epiploic artery transmit their vessels to the hilum of the spleen.

The lymphatic vessels draining into these glands pass very obliquely in the walls of the stomach.

We may therefore describe three chief lymphatic areas of the stomach.

(*a*) An area along the lesser curvature from which the lymphatic vessels pass upwards and to the left into the coronary glands.

(*b*) An area along the greater curvature from which the lymphatic vessels pass downwards and to the right into the glands of the greater curvature.

(*c*) An area, comprising the greater tuberosity of the stomach, extending up to the œsophagus, and on the greater curvature as far as the limit of the supply of the left gastro-epiploic artery.

If these statements are correct it would naturally be expected that in cases of malignant disease beginning in the pyloric half of the stomach the growth would extend in the direction of the curvatures, and that the glands along the curvatures would be affected, whereas the greater tuberosity of the stomach and the glands which drain it would escape. An examination of a large number of specimens bears out this supposition. Indeed, the third lymphatic area (*c*) of the stomach may be looked upon as an area apart, as one into which extension very rarely occurs, and as one, therefore, whose lymphatic vessels and glands remain healthy.

Upon the following grounds, then, I have based the operation which I describe below:

1. Malignant disease of the stomach commences most often near the pylorus, just below the lesser curvature.

2. From its point of origin it spreads most widely in the submucosa, and chiefly towards the cardiac end of the stomach.

3. On the duodenal side it spreads tardily, and never extensively.

4. The drift of the growth is towards the curvatures, chiefly the greater curvature.

5. The lymphatics draining the affected area pass to glands situated along the coronary and pyloric arteries above, and the right gastro-epiploic and the gastro-duodenal arteries below.

6. The greater tuberosity of the stomach is practically never involved by the spread of growth from the pyloric end.

In order to ensure complete removal of the primary growth, of the infiltrated lymphatic vessels, and of the glands into which those vessels drain, it will be necessary to remove the stomach as far up on the lesser curvature as the point of abutment of the coronary artery, and on the greater curva-

ture as far as the termination of the right gastro-epiploic artery, and to remove the first portion of the duodenum. This must be done in all cases, and unsparingly, if our results are to show improvement.

The necessary corollary of this requires, perhaps, more forcible emphasis. It is that the part of the stomach which the disease does not tend to involve should be left. The great tuberosity of the stomach is rarely invaded, and should therefore rarely be removed. Surgery should be as radical as is necessary, but should also be as conservative as is possible and safe.

The operation is carried out in the following way:—The abdomen is opened about half an inch to the right of the middle line through the rectus muscle, the fibres of which are split. The incision must be of good length, so that adequate room is afforded for the subsequent manipulations. The stomach being exposed, a preliminary ligature is applied to its chief vessels—the coronary, the pyloric, the gastro-duodenal, and the left gastro-epiploic. The veins running with the arteries are included in the ligatures, and in each case two ligatures are tied, and the vessels are divided between them. The coronary artery is exposed first, as it lies in the "ligament of Huschke"—the falx coronaria—whose folds enclose it. This is readily done if the position of the artery be defined by lifting up the liver and exerting traction upon the stomach, pulling the viscus forcibly downwards and outwards until a ridge is raised up in the peritoneum by the underlying vessel. The artery and vein are then surrounded by two ligatures, passed with an aneurysm needle or Greig Smith's forceps, and divided. If the vessel is easily seen it should be divided after the œsophageal branches have been given off, as these offsets carry blood not only to the œsophagus, but to a portion of the fundus of the stomach.

The pyloric artery and the gastro-duodenal are best found by exposing the hepatic artery, from which they both arise, separately or by a common trunk. The hepatic can be found a little above the pylorus before it passes upwards in front of the foramen of Winslow. A gentle traction of the pylorus downwards and to the right will render its discovery an easier matter, or the vessel may be ligatured after section of the stomach on the left, as suggested by Cunéo. The pyloric portion of the stomach, after section, is turned over to the right and dragged firmly. In the pancreatico-duodenal recess the vessels can be felt and safely ligatured. The left

gastro-epiploic is ligatured at the point of section of the stomach, at the junction of the great tuberosity with the body of the viscus. This point has to be guessed at approximately, but a little experience of stomachs in the *post-mortem* room will enable the operator to obtain the correct point accurately. The vessels being ligatured the omenta are separated from the stomach, and are divided at such a distance from the viscus as will ensure that all the glands remain with the stomach. A few points may require clipping and ligaturing during this manipulation.

It is important to remember that the middle cœliac artery is readily wounded in dividing the great omentum. Its injury

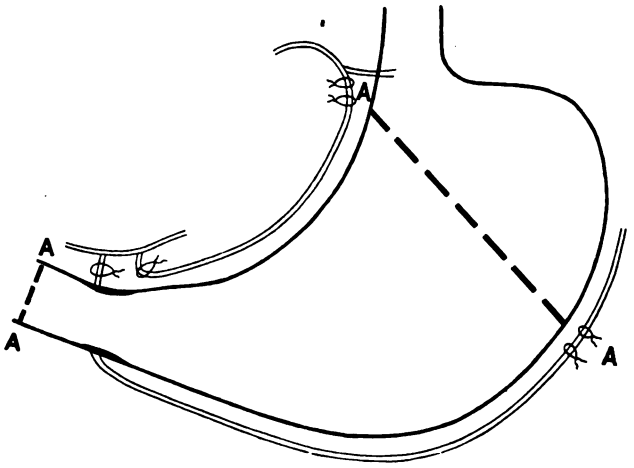

FIG. 2—Showing the ligatures applied to the vessels and the lines of section of the stomach and duodenum. *A A*, lines of incisions.

involves a risk of gangrene of the colon, and extreme care should therefore be exercised in dividing the omentum in its neighbourhood. The stomach and duodenum are now free. A Kocher's clamp is applied at the cardiac end, a Doyen's clamp on the duodenum, at the junction of the first and second portions, and the portion between the clamps removed. The portion taken away consists of the lymphatic areas *a* and *b*, the first portion of the duodenum, and all the lymphatic glands along the coronary and right gastro-epiploic arteries. After its removal a gland or two from the head of the pancreas may have to be dissected away, and possibly a small

portion of the pancreas itself may need removal, as in cases recorded by Richardson, Obalinski, Christy Wilson, and myself.

The anastomosis between the stomach and intestine must now be made. End-to-end approximation (Kocher's method) or a separate gastro-enterostomy with suture of the stomach and duodenal cut surfaces may be performed. In my own case I found it impossible to perform end-to-end suture, and I therefore closed both ends and performed a posterior gastro-enterostomy, pulling a loop of the jejunum up through a slit in the transverse mesocolon. End-to-end approximation is the most desirable method, and has the support of Mikulicz, Krönlein, and Terrier. If, however, the first portion of the duodenum is removed it will be found a matter of great difficulty, if not an impossibility, to effect an anastomosis without undue tension.

VI.—*Two Cases of Intra-cranial Section of the second and third divisions of the Trigeminal Nerve for Severe Neuralgia.* By THOMAS H. MORSE. *Read November* 22, 1901.

DURING the last ten years many valuable articles have appeared in the different journals on the surgical treatment of trigeminal neuralgia. In 1891 Mr. Victor Horsley published, in the *British Medical Journal,* three lectures. He there described an operation for removing the Gasserian ganglion, after having removed the bone over the temporal fossa and opened the dura mater over the temporo-sphenoidal lobe, so that the trunk of the fifth nerve could be divided between the Gasserian ganglion and its origin from the side of the pons.

In 1892 Mr. Rose, in the Lettsomian Lectures, gave a very full account of our knowledge of this subject, entering into the pathology, clinical history, and operative treatment, showing that reunion of the divided nerves was the common cause of failure to cure. This led him to divide the nerves as high up as possible, and finally to remove the Gasserian ganglion, which was reached by trephining the base of the skull through the pterygoid region.

In 1892, also, Hartley in America, and Krause in Germany, simultaneously described an intra-cranial but extra-dural method, a procedure now known as the Hartley-Krause operation, which has since been followed by Mr. Jonathan Hutchinson, jun., Mr. Lynn Thomas, and other surgeons.

In the two cases that I wish to bring before you also this method was adopted.

It is evident that to obtain a cure section of the nerves must be above the diseased part, hence the Gasserian ganglion has been removed, and even in Mr. Horsley's cases the trunk of the nerve was divided between the ganglion and its origin from the side of the pons; but the complete removal of the Gasserian ganglion necessitates tearing of the cavernous sinus, thus adding to the severity of the operation.

Now in many cases it appears that the disease is of peripheral origin; hence, in these cases, if section of the nerves

on the distal side of the ganglion can be performed and reunion prevented, a cure should be expected.

At the meeting of the British Medical Association at Portsmouth, in 1899, Mr. Lynn Thomas showed photographs and gave histories of cases in which he had divided the second and third divisions of the fifth nerve by the Hartley-Krause method, and to prevent reunion had blocked the foramen rotundum and foramen ovale with Horsley's wax, which was left in the skull without doing any harm. If it can be proved in course of time that the wax is not absorbed, and that reunion is permanently prevented by it, I think the removal of the ganglion or the section of the nerve above it will be unnecessary.

The two cases I wish to bring before you are almost exactly similar.

Case 1.—Mr. S., æt. 73, was referred to me by Dr. Burton-Fanning in August, 1899, with the following history:—For the last two years he had suffered from agonising paroxysms of pain, always occurring on the right side of the face over the area corresponding to the distribution of the second and third divisions of the fifth nerve. Most of his teeth had been removed without having any effect upon his condition.

The paroxysms occurred suddenly and were very acute. During their duration, which was for about a minute, he appeared as if suddenly stabbed through the jaws. He would call out and roll about holding his face; of late the pain has increased in severity and in frequency, occurring sometimes every ten minutes.

The movements of the tongue in the mouth will generally bring on a paroxysm, hence he is afraid to eat and is losing flesh in consequence.

He generally sits in his chair with the saliva dribbling from his mouth, being afraid to make any effort to swallow it. He has also acquired the habit of constipation from fear of the least straining at stool.

The quantity of urine passed in twenty-four hours has diminished to 10 oz., entirely due to his being afraid to swallow sufficient liquid. By careful feeding, however, this was increased during the few days that he was under observation before operation to two and a half pints.

Operation.—On August 24, 1899, ether was administered by Mr. Muriel. I was assisted, with his usual skill and

common sense, by the late Mr. Waring, to whom much of the successful issue of the case is due.

An incision was made down to the bone, commencing close to the external angular process of the frontal bone, along the temporal ridge, and down to the insertion of the ear. Having turned down the skin flap and cut away the greater part of the temporal muscle, I commenced with hammer and gouge to remove the bone, cutting thin shavings parallel to the dura mater until the latter was exposed sufficiently to get beneath it a strong pair of cutting gouge forceps, and with these rather more than two square inches of bone were removed. The dura mater was then easily detached from the floor of the middle fossa, and, together with the brain, pressed upwards. There was a constant oozing of blood, which obscured the field of operation. The second division of the fifth nerve came first into view passing into the foramen rotundum; this was caught up with a squint hook and divided with scissors; next the large trunk of the third division was found and divided; the Gasserian ganglion could be demonstrated with the large cavernous sinus lying directly above it, and quite intact.

I next put in a large plug of Horsley's wax for the two purposes of stopping hæmorrhage and of interposing a thickness of about a quarter of an inch between the divided ends of the nerves to prevent their reunion. The dura mater was then allowed to fall back into its place and the skin sutured.

The shock following was not great, and all pain ceased immediately after the operation.

He recovered completely, gained flesh, and lost his distressed look. It is now more than two years since the operation, and he has never had any return of pain.

The photograph shows the portion of bone removed; pulsation is still to be seen and felt over this area.

Case 2.—Mrs. C., æt. 66, sent to me for operation by Dr. Fanning.

History.—For the last ten years she has suffered from severe neuralgic pains on the right side of the head and face, over the parts corresponding to the distribution of the second and third divisions of the fifth nerve.

The pain has of late been more severe and more continuous. It comes on in paroxysms, which can be produced by any sudden noise, or any slight irritant such as a draught

of cold air, or even by a light touch. Both eating and drinking will cause it, hence she has lost flesh of late, not having been able to take sufficient food. Her sleep, also, is generally very disturbed; nearly all the teeth have been removed, the effect of which was only to increase the pain.

The pain was severe in the temporo-malar and auriculo-temporal nerves, also in the side of the tongue and in both upper and lower jaws of the right side, as well as in the skin covering them.

The first division of the nerve was not affected.

October 25, 1901.—Chloroform was given by Dr. Fanning, the head having been prepared antiseptically. I made an incision from the external angular process of the frontal bone, curving upwards along the temporal ridge and downwards to the highest part of the attachment of the ear.

The skin flap, together with the temporal muscle and as much as possible of the periosteum, was detached and drawn downwards. The bone was then cut with a mallet and gouge in such a way as to take off thin shavings of bone over a surface of about two square inches, always cutting parallel with the dura mater, until the latter was exposed sufficiently to insert the gouge cutting forceps, with which all the rest of the bone was removed to the required extent.

Next the dura mater was detached from the middle fossa of the skull, and together with the temporo-sphenoidal lobe pressed upwards and held so with a broad retractor until the Gasserian ganglion came into view, with the cavernous sinus lying upon it. The third division was easily seen and was caught up on a squint hook and divided with scissors; the second division of the nerve was then caught up and divided in the same way. During all this time there was a constant welling up of blood, and the various steps of the operation had to be performed in the intervals that were obtained by stuffing the wound with gauze, and waiting about a minute before removing it, and repeating this process when the field of operation was again obscured. I then put in a piece of Horsley's antiseptic wax and pressed it into the bottom of the fossa and into the foramen ovale and foramen rotundum, so that it remained about a quarter of an inch thick and spread out to about the size of a shilling, thus interposing a considerable plug between the divided ends of the nerves with the hope of preventing their reunion permanently.

DESCRIPTION OF PLATE III.

To illustrate Mr. Morse's two cases of Intra-cranial Section of the Second and Third Divisions of the Trigeminal Nerve for Severe Neuralgia.

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The wound was then united and recovery was uninterrupted. Pain ceased immediately after the operation, and up to the present time has never recurred; the patient is quite well.

I wish to call attention to the simplicity of this operation as compared with other methods, also to the rapidity with which it can be done, occupying on the dead subject from ten to fifteen minutes only.

VII.—*Five Cases of Operation on the Gasserian Ganglion for Epileptiform Neuralgia of the Fifth Nerve.*

By J. Hutchinson, jun. *Read November* 22, 1901.

ALL these cases were examples of inveterate and most severe neuralgia occurring in elderly subjects, four of them males and one female. Particulars of some of them have already been published in the *British Medical Journal* of November 5, 1898, and the *Transactions of the Medical Society*, vol. xxiii, 1900, p. 274, and the following brief summary will here suffice.

Case 1.—Thomas B., æt. 57, right fifth nerve affected for fifteen years. Many operations on branches of the second and third divisions, including neurectomy of the inferior dental and removal of Meckel's ganglion; pain returned worse than ever with intervals of only a few minutes between each attack; reflex spasm of facial and jaw muscles during the attacks; pain only alleviated by large doses of morphia. Excision of lower part of Gasserian ganglion, with second and third divisions, October, 1897 (Hartley-Krause method); bleeding so troublesome that operation had to be completed in two stages. Patient shown November, 1901, four years after operation; he has been perfectly free from pain ever since it was performed.

Note.—Before operation the neuralgia had involved the scalp as far back as the occipital region, though always much the worst in the distribution of the two maxillary nerves.

Case 2.—Thomas S., æt. 44. Duration of neuralgia fifteen years. In 1897 was operated on by another surgeon by the pterygoid route (Rose's method). The operation was done in two stages; it was very prolonged, and owing to hæmorrhage and shock his life was endangered. The Gasserian ganglion was thought to have been excised, but the neuralgia recurred after a short interval; subsequently he took to injecting morphia in very large amounts to relieve the pain. Operation by the temporal route April, 1899;

hæmorrhage considerable; external carotid artery ligatured; lower part of ganglion and second and third divisions removed. The aperture in the base of the skull at the prior operation was found to have been made a third of an inch outside the ganglion, which was quite intact. The patient, shown November, 1901 (two years and a half after operation), has been entirely free from pain ever since, and has not used a single dose of morphia.

Case 3.—Louisa C., æt. 56. Duration of neuralgia many years; no previous operation. Gasserian ganglion removed, with exception of ophthalmic division, September, 1898. Has greatly improved in health, and remains free from pain, November, 1901 (over two years).

Case 4.—Thomas W., æt. 64. Duration of neuralgia four years. Ophthalmic division involved, and pain felt during attacks in less degree over the whole right side of head. Operation June, 1899. Dura mater was thin, and tore readily. Whole of ganglion, with ophthalmic division, removed. Owing to pressure at the time of operation by the retractor, he had temporary left hemiplegia, which was ultimately quite recovered from. After some months, during which the right cornea had been sound though anæsthetic, an ulcer formed on it, which resisted all treatment, and finally necessitated removal of the eye. The patient (shown November, 1901) has remained quite free from pain since the operation (nearly two and a half years).

Case 5.—Samuel G., æt. 64. Neuralgia for some time confined to the inferior maxillary division, which was operated on extra-cranially by Mr. Stanley Boyd. In August, 1891, I performed neurectomy of the inferior dental nerve by trephining the lower jaw; relief for several months, then recurrence in both lower divisions of the fifth. Operation on Gasserian ganglion (by temporal route) October 7, 1901. In this case the middle meningeal artery was not ligatured; in the four previous ones this vessel was tied and divided. The patient was shown November, 1901, as an illustration of the absence of any deformity produced by the operation.

It will be noted that in only one of the five cases was the ophthalmic division removed, yet in all of them there has not

been the slightest return of pain. It is thought by some that if the lower part of the ganglion and its two main divisions are excised the neuralgia may return in the first division. I do not think this has been proved in any case, although, of course, it is possible. On the other hand, it must be remembered that in four out of five cases of severe epileptiform neuralgia the ophthalmic division is not involved, and it is obviously a great advantage if it can be spared by the operator. For in the first place, whatever precautions are taken after the operation, an anæsthetic cornea is liable at any time to become ulcerated; this was illustrated by Case 4, and is no doubt due to the access of septic dust which is not felt and is allowed to adhere to the cornea. In the second place, the chief difficulty and risk of the operation are met with in dealing with the upper edge of the ganglion and the ophthalmic division. Many cases have been recorded in which the fourth, the sixth, and even the third nerve have been damaged at this stage, or the cavernous sinus opened. At present there is no doubt that the operation by the temporal route is by far the best, and it is not too much to say that the various methods of approaching the ganglion from below with their drawbacks of division of the zygoma, scarring of the face, etc., should be entirely discarded. Their chief defect is that the ganglion cannot be satisfactorily exposed by means of them. The combination of the two methods known as Doyen's operation has no advantage whatever, and it should be noted that two out of three of Doyen's own cases died within ten days of the operation. There is no necessity to make a large flap on the side of the head, the base of the flap corresponds to the upper border of the zygoma, starting an inch behind the external angular process, and finishing above and behind the condyle of the lower jaw. The bone removed is the thinnest part of the cranial vault, and a large trephine followed by Hoffmann's cutting forceps seems the best instrument for the purpose. I have not replaced the bone in any of my cases. If it is possible to avoid division of the middle meningeal artery, as is sometimes the case, this is certainly an advantage. The constant oozing from the dura mater which is so troublesome may be largely avoided by keeping the patient in the sitting posture. The suggestion made in America that the dura mater should be deliberately incised, and the cerebro-spinal fluid let out, is, I am sure, a mistaken one. In the only case of the five now reported in which the dura mater was opened, the pressure

on the brain caused by the retractor was followed by temporary hemiplegia. The escape of cerebro-spinal fluid for some days after the operation must be both a disadvantage and a danger. An electric head lamp is almost essential to enable the ganglion to be properly and cleanly dealt with. In several of the reported cases (Tiffany, *Trans. Amer. Surg. Assoc.*, 1896) the operator is supposed to have "broken up the ganglion" with a scoop. It need hardly be said that such a proceeding must be as futile as it is dangerous.

The mortality following the operation.—Tiffany (*loc. cit.*), in 1896, collected 108 cases, including nine of his own, and the mortality of these worked out at 23 per cent. Commenting upon this heavy mortality Ransohoff stated his belief that the true mortality of the operation would be found to be nearly 50 per cent. Such an estimate must be entirely beyond the mark, and even Tiffany's record, with a death rate of one in four, cannot be accepted as representing the real danger of the operation in competent hands. His 108 cases had been operated on by no less than forty-seven surgeons, and by such varying methods as the Hartley-Krause one, intra-dural section of the root, Rose's method, Doyen's, and the like. It is certain that the mortality following excision of the Gasserian ganglion by the temporal route ought not to exceed from 5 to 10 per cent. It should steadily diminish with increasing experience of the operation, a resort to it before the patient is worn out by his suffering, and by the adoption of the method described in which the ophthalmic division is spared. Besides the five cases mentioned above I have operated on the Gasserian ganglion in one other, but in the last a tumour was found growing from the base of the skull and pressing on the nerve; this could only partially be removed, and the relief afforded was only temporary.

VIII.—*A Case of Partial Excision of the Gasserian Ganglion.* By CUTHBERT S. WALLACE. *Read November* 22, 1901.

H. E., male æt. 31, a wheelwright. The neuralgia began six years ago, affected the second division, and was often brought on by movements of the tongue or mastication. Two years after commencement of the illness the infra-orbital nerve was divided at the foramen. The procedure gave him relief for three weeks only. Latterly the pain had become worse, so that hardly a night passed without an attack, and any attempt to follow his occupation was prevented by a severe attack of tic. Patient was admitted under Dr. Box into St. Thomas's Hospital, who, after trying all remedies, advised the patient to submit to operation. The greatest number of attacks in one day was twenty, though they were usually of short duration. The mortality of the operation was put to him, and he decided to run the risk.

The Hartley-Krause route was chosen, and from the outset the hæmorrhage was very free. In approaching the ganglion the middle meningeal was torn, and as the bleeding was very free and could not be stopped, the external carotid artery was tied, and the operation proceeded with. The second and third divisions and the ganglion were exposed, but in the separation of the ophthalmic division a sinus was wounded, and exceedingly free bleeding was the result. As this could not be controlled and a proper view obtained at the same time, the second and third divisions with the corresponding part of the ganglion were cut away, and the wound plugged and then closed. (Date September 11, 1901.) Patient since that time has been free from pain. The area supplied by the ophthalmic division is practically normal, the anæsthesia corresponding to the usual distribution of the second and third divisions, including half the tongue, palate, and inside of the corresponding cheek.

The part of the ganglion removed has been kindly examined by Dr. Seligmann, who states in a provisional report that there are large numbers of nerve-cells, some normal, and others in various stages of degeneration, as shown by disarrangement of the Nissl bodies.

Whether this method of dealing with cases of facial neuralgia will prove useful depends on the relief afforded. If this is permanent, then the operation is certainly to be preferred to the more severe operation with its danger to the eye. Tiffany, in a very exhaustive paper, published in the *Annals of Surgery*, 1896, advocated this method and laid great stress on the fact that the portion of the ganglion corresponding to the nerves must be removed, as simple intra-cranial section of the nerves was extremely likely to be followed by a recrudescence of the pain.

IX.—*A Case of Fragilitas Ossium.* By Warrington Haward. *Read November* 22, 1901.

P. E., a boy æt. 10, was admitted under my care into St. George's Hospital, on October 18, 1899, on account of severe deformities of the lower limbs rendering him completely unable to stand or walk. He was a bright, intelligent boy, rather pale and thin, with dark hair and eyes. His head looked somewhat large, but this was only because the body and limbs were small for his age. The skull showed none of the characters of rickets. He sat upright, and there was no spinal curvature. The muscles of the head, trunk, and upper limbs were fairly well developed, and were used actively; those of the lower limbs were wasted and feeble, especially those on the anterior surface of the legs. The chest was slightly compressed laterally, and the sternum rather prominent, but he had good use of the muscles of respiration.

The upper limbs showed far less marked changes than the lower. The bones of the arm and forearm were of fair length, but thin and narrow, and all of them showed a moderate degree of outward curvature, and numerous slight irregularities due to old fractures. The hands, in respect to both bone and muscle, were well developed.

The bones of the lower limbs were all thin and small. The femora showed a moderate degree of curvature, both outwards and forwards, besides the irregularities marking former fractures.

The most striking deformities were those of the legs. The tibia and fibula of each leg were extremely thin and narrow, flattened laterally, with very sharp margins, and a marked forward curvature. The feet were thin and small, and much less developed than the hands.

The curvature of the bones of the right leg was extreme, so that the lower half of the leg formed a right angle with the upper half; the tendo Achillis was tense, and the skin cicatricial and adherent over the most prominent part of the skin. The bones of the left leg were similarly curved, though to a rather less degree.

The photographs, kindly taken by Mr. Clinton Dent and Mr. H. G. Drake-Brockman, show well the condition of the limbs, and the skiagrams taken by Mr. F. T. Addyman give an accurate idea of the state of the bones. The heart and lungs appeared to be healthy. The bowels acted regularly and the evacuations were natural. The temperature was normal. The urine presented striking deviations from that of health. It looked natural when passed, but on standing deposited a large quantity of thick phosphatic sediment. Mr. J. Addyman Gardner kindly furnished me with the following analysis:

Specimen of urine, October 28, 1899.—Sp. gr. 1010.

Lime: CaO per 100 c.c. = ·4478 gr. or ·4433 per cent. by weight. Phosphates: P_2O_5 per 100 c.c. = ·0183 gr. or ·018 per cent. by weight; from which it will be seen that the urine contains a great excess (about twenty-five times the normal) of lime salts.

This boy is the seventh child of healthy parents, and the only notable point in the family history is the rapid pregnancies of the mother. He has been well fed and taken care of, and has lived in a healthy part of Surrey.

The mother married at the age of twenty. She had two miscarriages (at between the third and fourth months); then six children were born in the ensuing six years; after this there were three miscarriages, and then this boy was born. The confinement was natural, but during this pregnancy the mother was weak, and could not eat meat. After this boy there was no pregnancy for three years, and then another boy was born. All the other children, with the exception of one, who died at the age of five months of some throat affection, are living and healthy. Immediately after birth it was discovered that this boy had both humeri fractured, as well as two ribs on the right side. Since then twenty-three distinct fractures have been noted, mostly from very slight causes. The bones have always united without difficulty, but when about three years old the legs began to be curved.

He was never able to walk alone, but when about two years old could stand with assistance, and would occasionally walk a few steps holding some one by the hands, or resting upon a table or chair. Fractures of the bones of the legs, however, became so frequent that any attempt at walking was soon abandoned.

During the first two years of infancy his spine easily bowed,

and his head dropped forwards, and for this reason he wore a spinal jacket, but since the age of three or four he has been able to sit upright without support.

He was not suckled after the first week, the mother being then too weak, and having but little milk; but there was no difficulty about his feeding. He digested cow's milk easily, and was fed, at first, on that and Mellin's food. Subsequently he was given meat-juice and other nourishing food, of which he took plenty, and with good appetite and digestion. He has not suffered from any material illness, and since infancy he has been fed like other children, excepting that special care was taken to make his diet as nourishing as possible.

As soon as the boy had been carefully examined and an analysis of the urine obtained, he was given cod-liver oil and steel wine, together with a liberal diet, including meat, potatoes, and green vegetables, eggs, milk (three pints daily), and beef-tea.

An attempt was made to straighten the legs by weight extension, but without effect.

January 2, 1900.—In making some movement in bed his left leg became bent under him and the femur fractured near its middle. The limb was treated by splints and vertical extension, and the bone united well in six weeks.

March 10, 1900.—Mr. Gardner kindly made another analysis of the urine, which showed the excretion of lime to have greatly diminished, viz. from ·4478 gr. to ·0599 gr. per 100 c.c. This urine on standing yielded no deposit such as occurred in the previous specimens, and evidently pointed to a great diminution in the waste of bone material.

This improvement in the condition of the urine and the good general health of the boy encouraged me to attempt to remedy the deformity of the legs by osteotomy.

On March 27 I operated on the right tibia. The periosteum was carefully stripped back over the most prominent part of the curve, and a wedge-shaped portion of bone removed of sufficient size to allow of the bone being brought to a straight line; the ends of the divided bone were then wired together and the periosteum replaced. The tendo Achillis was also divided. The fibula, which was extremely thin, was fractured, but no part of it removed.

The limb was placed in a straight splint.

The wound healed at once, and the bones quickly united. The wire was removed at the end of three weeks.

June 1.—A similar operation was performed on the left tibia, with a corresponding result.

The legs were put up in a silicate splint for another month, and then a laced leather splint was applied, which was daily removed for gentle massage, and on August 16 he was taken home. He could then move his legs freely, but had not attempted to walk.

The urine was quite natural.

The bone where sawn was very hard. The piece removed was examined microscopically, and a section showed no notable change from normal bone.

I am indebted to Mr. Gardner's kindness for the following analysis of a portion of the bone removed:

Moisture	14·78	
Phosphoric acid (P_2O_5)	22·53	= 49·18 per cent. of calcium phosphate.
Calcium oxide	29·68	
Sand	0·97	
Organic matter, CO_2, and volatile salts	29·21	
Alkalies, iron oxide, etc.	3·73	
	100·00	

This shows a deficiency, but not a very great deficiency, of calcium phosphate, as compared with healthy bone (about 58·00).

But it will, of course, be remembered that the specimen analysed was taken from the bone when the boy had been under treatment for several months, and when the urine had ceased to excrete the large quantity of lime which had formerly been passed.

On February 1, 1901, six months after he left the hospital, his mother wrote to me that he had fallen off a chair and bruised his legs. "But," she added, "his bones must be very much stronger, as before if he had fallen he would have broken them. . . . He is very well, and looks bonny." And on October 7, 1901, I had another letter saying that he can stand with assistance and put a little weight on his legs, and that he is learning to walk between two poles.

I do not gather, however, that he has made much progress in this direction, the leg muscles still remaining very feeble and wasted.

A case greatly resembling this, but in an older patient (a man æt. 29 years), was exhibited to the Medical Society by Mr. Clinton Dent in 1896. (For description of the case, with photograph and skiagraph, see *Transactions of Medical*

Society of London, vol. xx, p. 339.) The deformity of the legs in Mr. Dent's case was almost exactly like that of my patient, and there was a similar condition of urine—"much phosphatic deposit, at times forming an almost mortary sediment."

What is the cause of this curious error in nutrition, whereby the bone salts are excreted in the urine, and the bones remain ill-developed, soft, and of extreme fragility, I do not know.

In my case fractures were observed immediately after birth, and in Mr. Dent's case at the age of four months, so that the condition could not have depended upon the diet of the children.

It is noteworthy that the upper limbs, which were used, exhibited comparatively little abnormality. The bones were thin, and gave evidence of numerous fractures, but the degree of curvature was in striking contrast to the condition of the lower limbs. The legs, which he had not used since the slight attempt at walking in his second year, showed extreme curvature, the posterior muscles having drawn the heel with the lower half of the tibia backwards and upwards. The thighs, the muscles of which were necessarily more used in the movements of the body, showed correspondingly less deformity.

The only suggestive points in the history which I was able to obtain were the rapidly succeeding pregnancies of the mother, and her weakness before this child was born. Very notable, however, was the change in the condition both of the urine and of the bones in the five months before the operation, during which the boy was taking a very nutritious diet, and also cod-liver oil.

DESCRIPTION OF PLATE IV.

To illustrate Mr. Warrington Haward's case of Fragilitas Ossium.

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DESCRIPTION OF PLATE V.

To illustrate Mr. Clinton Dent's case, referred to in Mr. Warrington Haward's paper.

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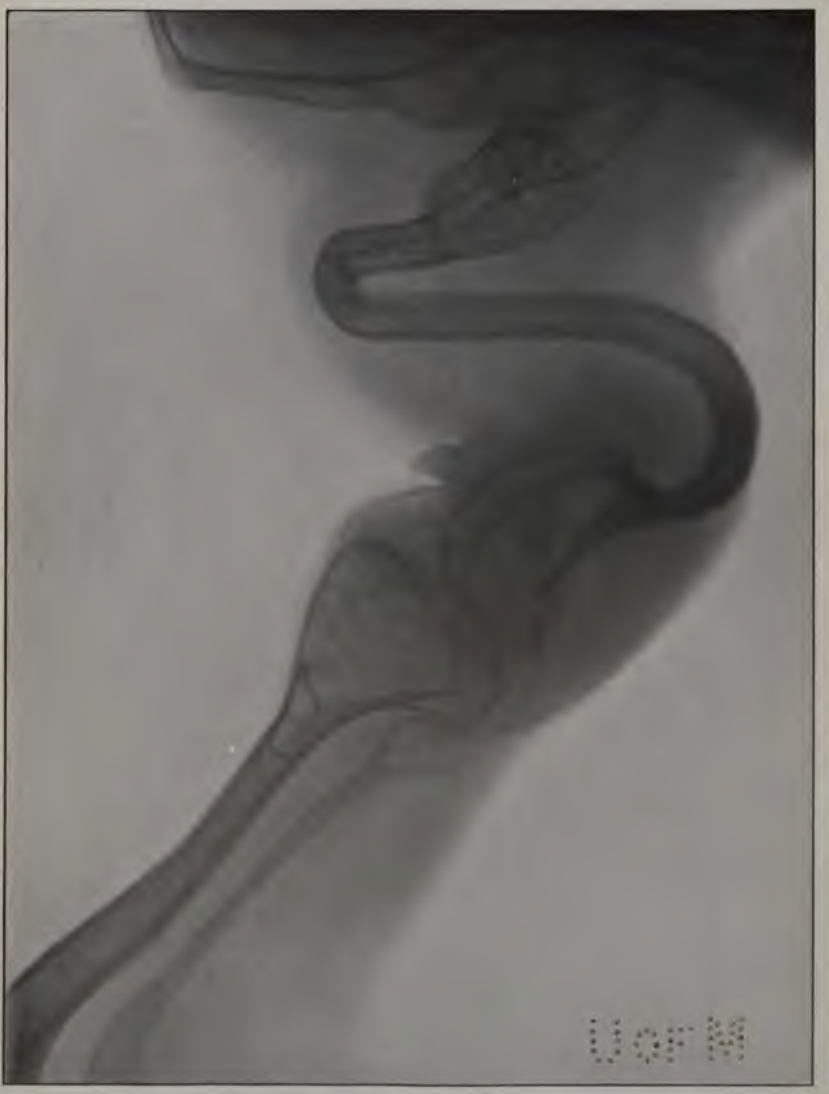
NOTICE

DESCRIPTION OF PLATE VI.

To illustrate Mr. Clinton Dent's case, referred to in Mr. Warrington Haward's paper.



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X.—*On the Theory of Compensation in Incompetence and Stenosis of the Mitral Valve.* By T. Stacey Wilson, M.D. *Read December* 13, 1901.

THE object of this paper is to make a clinical contribution towards the proof of what may be called the modern theory of compensation in disease of the mitral valve.

For the last fifteen or sixteen years the belief has been gaining ground that the filling of the left ventricle of the heart during diastole is mainly due to aspiration by the ventricle itself rather than to the contraction of auricle or other forces.

This view as to the filling of the ventricle, which is based upon observations such as those of Dr. H. D. Rolleston (who demonstrated the presence of a negative pressure in the ventricle during diastole), has, since 1885 or earlier, been advocated by some writers and lecturers on cardiac physiology. A very clear statement of this theory and its bearings upon the functions of the auricles will be found in some *Lectures on the Physiology of the Vascular System,* by Mayo Collier, published 1888.

But although the physiological side of this question has for so many years been clearly stated, the new views as to ventricular aspiration do not seem to have been generally adopted by clinicians, and the necessary modifications in the theory of compensation in mitral disease have very rarely been faced in the literature of the present day.

Amongst the exceptions must be mentioned the name of Dr. Clifford Allbutt, who accepts the theory in his *System of Medicine.*

The new theory seems to be much more satisfactory than the old for many reasons. It is more satisfactory to believe that the strong left ventricle is able to compensate for its own deficiencies instead of having to fall back for assistance, as the old theory would have us believe, upon the weak right side of the heart, or the still weaker pulmonary capillaries. Also from clinical and pathological standpoints it proves more satisfactory than the old one, and I have adopted it as a

working hypothesis for the last seventeen years, and have not yet found it to be at fault.

The facts brought forward in this paper are the outcome of an investigation into the loudness of the heart-sounds in health and in valvular disease, but more especially as regards the loudness of the second cardiac sound in the pulmonary area.

According to the old, or *vis-a-tergo* theory of compensation, the pulmonary second sound ought to be always more or less accentuated in mitral disease, because a very considerable portion of the work of compensation is supposed to rest upon the right ventricle. According to the new, or *vis-a-fronte*, theory almost the entire work of compensation is done by the left ventricle itself, owing to an increase in its aspiratory power, and therefore, according to this theory, when compensation is good the right ventricle will have no extra work to do, and the pulmonary second sound will be of normal loudness.

The result of my observations points unmistakably towards the accuracy of the latter, or *vis-a-fronte*, theory.

For the purpose of measuring the heart-sounds I found, after many experiments in obliteration of the heart-sounds by the interposition between the stethoscope and the chest-wall of discs of rubber, felt, sponge, etc., that the simplest and best method was to use a bag of air for cutting off the heart-sounds. To do this an empty thin-walled, elastic rubber bag (a deflated toy balloon) is placed between the chest-piece of a binaural stethoscope and the chest-wall, and by its inflation the stethoscope is slowly lifted up until the heart-sounds are no longer heard. The height to which the particular sound is audible is measured by means of a small platform placed between the stethoscope and the bag which slides up and down on three legs, which are graduated in fractions of an inch, and upon which can easily be read off the height of the chest-piece of the stethoscope when the sounds are lost. The inflation can be done by the mouth through a rubber tube, which can be compressed by the lips or teeth when it is desired to maintain the fulness of the bag for a time. Very exact measurements can be quickly made by this apparatus. In the statistics which follow I have adopted $\frac{1}{16}$ inch as the standard for measuring the distance to which the sounds are audible, and in speaking of the loudness of the sound I shall only give the number of sixteenths of an inch at which it is still audible, *e. g.* a sound whose loud-

ness I call "sixteen" is one audible one inch away from the chest-wall with the apparatus used. A sound of "twenty-four" is one audible one and a half inches away.

The observations I have made have shown that this method of measuring the sounds is most reliable, and, moreover, removes some sources of fallacy. It enables the listener to distinguish between an accentuation of the second sound due to real increase in the loudness of the valvular sound, and one due simply to the normal sound being better heard owing to the pulmonary artery being more superficial than usual. Also in several cases where there was apparently well-marked accentuation of the pulmonary second sound, I found that the sound was, in reality, not audible more than the usual distance from the chest-wall, and therefore could not be said to be accentuated. In these cases it was sharper and more slapping than normal, but there was no true increase in the volume, such as might be expected if the blood-tension in the pulmonary artery were raised.

I have recorded the measurements in sixteenths of an inch, and in the tables at the end of this paper the numbers given tell the number of sixteenths of an inch from the chest-wall at which the particular sound is audible.

In the course of the last few months I have made over 100 observations of all the heart-sounds in some fifty patients suffering from various ailments, and in the cases of mitral disease, some twenty-six in number, I have paid special attention to the pulmonary second sound, making some 150 observations of the loudness of this sound alone.

From these observations it appears that the pulmonary second sound varies in healthy hearts from 10 or 12 up to 20, and that slight ailments or a little excitement will increase its loudness up to 20 or 24, or more.

In anæmia in young women, on the other hand, the loudness of the pulmonary second sound is apt to range much higher. This is, no doubt, partly due to the very superficial position of the pulmonary artery in these cases, but there must also be some true accentuation. Amongst some four or five cases which I have examined, I have in one case recorded a pulmonary second sound heard $\frac{48}{16}$ inch from the chest-wall, and two others of 44 and 36 respectively.

In comparison with these very loud sounds of anæmia, heard as much as three inches (or $\frac{48}{16}$ inch) away from the

chest-wall, it is very striking to find that in disease of the mitral valve the pulmonary second sound can rarely be heard more than twenty-two to twenty-four sixteenths away (*i. e.* $1\frac{3}{8}$ to $1\frac{1}{2}$ inches), and in only one case out of the twenty-six examined was the pulmonary second sound regularly heard as much as twenty-six sixteenths away (*vide* Table II).

The fact that there is no very marked increase in the loudness of the pulmonary second sound during the process of compensation is also shown in Table III, which shows a series of observations upon a case of mitral stenosis with marked failure of compensation, who speedily recovered under treatment, and in whom the regular loudness of the pulmonary second sound never exceeded 22.

In one case of compensated mitral regurgitation the onset of anæmic dilatation of the right ventricle was accompanied by a rise in the loudness of the pulmonary second sound from 16 or 18, at which it usually stood, up to 44.

In another case a little nervous excitement caused a pulmonary second sound, usually heard only sixteen or twenty sixteenths of an inch away, to be audible at a distance of 28, and once at 40.

The discussion of the cause of this low average in the loudness of the pulmonary second sound would be out of place here, but the fact is interesting as showing that the pulmonary second sound is much louder in anæmia and nervous excitement than it is when the right ventricle is supposed to be working at its hardest, in order to try and overcome the embarrassment of the pulmonary circulation due to mitral stenosis or regurgitation.

I now turn to the theoretical consideration of the newer theory of compensation, in order that the bearings of my observations on the theory may be estimated; and I have, for the sake of brevity, expressed the theory in a series of propositions, some self-evident and some debatable, and needing any support which my observations as to the loudness of the heart-sounds can give.

In Mitral Incompetence.

Proposition I.—The process of compensation necessitates dilatation of the cavity of the left ventricle, as well as hypertrophy of its walls, and the amount of dilatation in the compensated heart is directly proportional to the amount of

blood which regurgitates into the auricle at each beat of the heart.

As mitral incompetence involves the leakage back into the auricle of a certain part of the contents of the ventricle at each beat of the heart, it therefore follows that in order to compensate for this leakage and allow of a normal amount being thrown into the aorta at each beat,—

1. Firstly, the ventricle must enlarge so as to hold both the normal amount and that which will escape back into the auricle.

2. Secondly, it must thicken its walls so as to be able to move this additional volume of blood.

3. The theory of the enlargement of the ventricular cavity is as follows:—On the first occurrence of regurgitation through the mitral valve a progressive over-distension of the auricle will theoretically result from the leakage of blood back into that chamber. As soon, however, as the size of the auricle exceeds that of the ventricle, the latter will, during its expansion, be drawing towards itself more blood than it can hold, and the momentum of the blood which cannot find entrance will tend to dilate the cavity of the ventricle. In this way the ventricle will be dilated by its own aspiratory force until its size is comparable to that of the auricle. This will not occur until the ventricle is large enough to hold the full amount of the leakage in addition to its normal contents (*vide* writer's paper, *Clin. Soc. Trans.*, vol. xxxiv, case 3).

Proposition II.—The dilatation and hypertrophy, which can compensate for the defective working of the left ventricle during systole, will, according to this theory, also relieve the auricle from embarrassment during diastole by ensuring the proper filling of the ventricle.

For (1) since the enlarged ventricle is able to accommodate all the regurgitated blood in addition to its normal contents, it follows that it will be as easy for the abnormal amount of blood to enter the abnormally large ventricle as for the normal amount to enter the normal ventricle;

And (2) the increased muscularity of the enlarged ventricle will ensure the slight addition to the aspiratory force which is required to draw the larger amount into the ventricle.

Proposition III.—Therefore, by the time the expansion of the ventricle has ceased, the auricle will have been relieved

of all the regurgitated blood, and only the normal amount will remain to be dealt with by it.

Proposition IV.—Therefore, in fully compensated mitral regurgitation no extra work will be thrown upon the left auricle, and there will be no embarrassment of the pulmonary circulation and no accentuation of the pulmonary second sound.

In Table IV are given the details of five cases of compensated mitral regurgitation, two of them, Cases 4 and 5, rather severe cases, and the others well-marked cases. In each of these it will be seen that the average loudness does not exceed seventeen, which is normal.

Proposition V.—Neither would there be any dilatation of the left auricle unless the regurgitation were extreme.

Since the systole of the auricle and of the ventricle together occupy one half of the cardiac cycle, it follows that only one half of the full volume of blood will normally enter the auricle during this portion of the cycle. Therefore there will be room in the auricle during the ventricular systole for any amount of regurgitated blood up to one half of the total quantity put into circulation at each beat. If, on the other hand, the amount of regurgitation were to exceed one half of the normal output of the ventricle there might be dilatation of the auricle, even when compensation was fully established.

Proposition VI.—According to this theory of compensation there ought to be a slight increase in the loudness of the aortic part of the second sound, because the greater expansile force of the hypertrophied and dilated ventricle will induce a more powerful recoil of the blood in the aorta, and will result in a more powerful closing and stretching of the aortic valves than in the case of the normally acting ventricle.

In Mitral Stenosis.

According to this theory, the left ventricle increases its aspiratory power in order to facilitate the entry of blood through the narrowed orifice, and so establish compensation. This is brought about as follows.

Proposition VII.—In mitral stenosis the left ventricle contracts with greater force than normal in order that the

rebound, both muscular and elastic, may be correspondingly increased.

1. An evidence of this is the increased loudness of the first sound which occurs in mitral stenosis.

This is shown in Table III, which deals with Case 3. Before the establishment of compensation the first sound is seen to be 32, sometimes reaching 40, and after compensation is established it is 22 or over, which is above the normal.

Very loud first sounds are also seen in Cases 7 and 8 of Table V and Case 10 of Table VI, which are severe cases of mitral stenosis. Also in Table VI the first sound in the five cases with good compensation averages 20, as compared with an average of about 12 in the five cases of mitral regurgitation.

2. Temporary embarrassment of the circulation is accompanied by an increase, often excessive, in the loudness of the first sound, which lessens again as the circulation improves (*vide* Tables III and V).

This is well shown in Case 7, Table V, where the loudness of the first sound is suddenly increased from 36, a sound inaudible with the stethoscope $\frac{1}{16}$ inch from the chest wall (if no conductor be used), to a sound audible without the stethoscope at a distance of two feet from the chest. The same increase to a less extent is seen in Cases 8 and 9, and is often noticeable.

3. This increase in the force of the ventricular contraction is also suggested by the occurrence of the hypertrophy of the left ventricle which is not infrequently noticed in mitral stenosis.

Proposition VIII.—The left ventricle alters its beat. Its contraction terminates with greater suddenness and force than normal. This concentration of ventricular effort at the termination of the systole has the effect of further increasing the force of the ventricular recoil which immediately follows the systole, lasting as it does only $\frac{1}{10}$ of a second.

1. This is the explanation of the sharp and loud end of the first sound which is so characteristic of mitral stenosis.

2. The characteristic cardiograms of mitral stenosis often show a more sudden recession of the heart from the chest wall at the end of the systole than in the normally beating heart.

Proposition IX.—As a result of this additional aspira-

tion the auricle can be relieved of much, if not all, of the additional work which the narrowness of the mitral valve entails. We find very frequently an absence of dilatation of the left auricle in mitral stenosis where compensation was perfect at the time of death (*vide* Dr. Samways, *British Medical Journal*, ii, 98, p. 365).

Proposition X.—Therefore in compensated mitral stenosis, if the narrowing be not very excessive, no extra work is thrown upon the right side of the heart, and there is no engorgement of the lungs.

1. As evidence of this, we find the pulmonary second sound of normal loudness, even in cases of severe mitral stenosis (*vide* Table VI and Table V, Cases 7 and 8, in part).

2. We find that the accentuation of the pulmonary second sound, which is characteristic of failing compensation, gradually lessens as compensation improves (*vide* Table II).

3. We may find no clinical evidences of pulmonary congestion or engorgement, even in severe cases of mitral stenosis.

Proposition XI.—We may also expect some increase in the loudness of the aortic second sound, apart from that due to increased arterial pressure, because of the increased ventricular aspiration, as suggested in Proposition VI.

In conclusion, although I am not able to bring a very large number of observations, owing to the amount of time required to make the careful observations here recorded, I nevertheless hope that I have brought forward enough evidence to show the desirability of making wider investigations with regard to this important question.

The importance of this question, so far as treatment is concerned, is obvious, for if the expansion movement does play an important part in compensation, we are greatly handicapped as regards the treatment of our patients by our almost entire ignorance as to the effect of drugs upon that important expansion movement. A recognition of our need of having cardiac tonics classified into those which increase the expansile force of the ventricle and those which do not will be the first step towards having that need supplied.

Summary of the Tables,

Showing the loudness of the heart-sounds in health and in valvular disease.

The figures given show the distance, in sixteenths of an inch, to which the sound is audible with the apparatus used (*e. g.* a pulmonary second sound of "16" is one audible at one inch distance, one of "40" is one audible two and a half inches from the chest wall).

	Pulmonary area.		Aortic area.		
	2nd sound.	1st sound.	2nd sound.	1st sound.	
I. Averages of—					
6 normal hearts . . .	16	14⅓	14½	11	} Table I.
4 normal hearts . . .	10	16	11	14	
5 hearts in slight anæmia, rheumatism, etc.	20	26	20	21	
3 cases of extreme anæmia, etc.	40	29	19	15	
II. Cases with compensated mitral regurgitation (average loudness [*see* Table IV]):					
Case 2 . . .	17	12	13	7½	9 observations.
Case 3 . . .	16	10	11	12½	6 ,,
Case 4 . . .	7	7	5	6	2 ,,
Case 5 . . .	16	18	22	15	5 ,,
Case 6 . . .	16	10	9	6	3 ,,
III. Cases with compensated mitral stenosis (*see* Table VI):					
Case 10 . . .	10	21	8	10	4 ,,
Case 11 . . .	14	19	8	13	2 ,,
Case 12 . . .	19	20	23	16	3 ,,
Case 13 . . .	13	20	14	20	2 ,,
Case 14 . . .	16	20	17	23	4 ,,
IV. Cases with failing compensation in mitral stenosis:					
Case 1 . . .	8	48 = ½″	14	36	1 observation.
Case 8 . . .	23	70 = ¾″	8	30	1 ,,
Case 7 . . .	24	2 feet	26	48	
Case 10 . . .	22	44	—	—	

Table I.

Table showing measurements of the loudness of the normal heart-sounds in the pulmonary and aortic areas respectively.

The figures given in this and the following tables are measurements in sixteenths of an inch of the distance from the chest wall at which the sound is audible with the apparatus used. Figures in brackets (16) are distances at which the sound in question is *sometimes* heard. Figures in square brackets thus [16] show the distance at which, in an irregularly beating heart, the sound may very occasionally be heard.

Sex.	Age.	Pulmonary area.		Aortic area.	
		2nd sound.	1st sound.	2nd sound.	1st sound.
F.	49	6	16, 18	10, 12	16
„	„	12, 9, 12	22 (24)	16	20 [24]
M.	51	10, 10, 12	14, 16	10	12 (16)
M.	36	10, 10	16, 16	8	9 (12)
„	„	9, 9	16, 16, 15	8	12
F.	19	16	10, 11 (12)	16	12
F.	23	12 (14)	16	14 (16)	14 (16)
F.	21	17 (22)	15, 20 [22]	14 (18)	12 [18]
M.	34	14	11	7	4
M.	41	16, 14	12	15	12
F.	44	16	20	15	12
F.	23	16 (18)	12 (18)	20	12 (14)
Average .		13½	16·2	14·3	12

TABLE II.

Table showing a classification of 150 observations upon the loudness of the pulmonary second sound in twenty-six cases of mitral incompetence and stenosis, according to their loudness, and also to their occurrence in patients with fully established compensation, or in patients treated for failing compensation. This second group includes observations made on these patients after the establishment of compensation. In the uncompensated group in some nine instances feeble sounds were due to feebleness of the ventricle and not to the establishment of compensation. For the sake of completeness I give in a second column, enclosed in brackets, the number of "occasional" observations made in addition to the regular ones.

Degree of loudness measured in sixteenths of an inch.	Number of observations made in fully compensated hearts.	Number of observations made in uncompensated hearts.
0 to 9	4 and (1)	2 and (1)
10 to 14	3 and (3)	18 and (2)
15 to 17	11 and (1)	9 and (2)
18 to 19	3 and (0)	10 and (0)
20 to 24	4 and (3)	37 and (19)
25 and over	0 and (1)	5 and (12)
	25 and (9)	81 and (36)

Table III.

Case 1.—Measurements of the loudness of the heart-sounds in a case of mitral stenosis with extreme dilatation of the right ventricle and dropsy, showing the variations taking place during the restoration of compensation.

On the date of the first measurement the relative cardiac dulness extended two inches to the right of the sternum in the fourth interspace, and the apex was in the sixth left interspace some five inches from the sternum, *i. e.* in the anterior axillary line. At the time of the last measurement the apex was in the fifth left interspace, four inches from the sternum, and the patient was walking about with very little dyspnœa.

Date.	Pulmonary area.		Aortic area.		
	2nd sound.	1st sound.	2nd sound.	1st sound.	
23, viii	8	32 (40) [48]	14	36 (24) [44]	Very ill.
25, viii	22, 22	24 (28)	20, 21	22 (28)	Much better.
26, viii	20 (22) [32]	22 [32]	16 (20)	28	Improving.
28, viii	16 [17]	22	10 (12)	22	,,
4, ix	18 [22]	22	16, 16	22	,,
,,	20	—	18	—	,,
20, ix	20 [22]	24	20	22	Got up on the 18th.
29, ix	20, 19 (22)	22 (24)	25	25	
,,	22 (24)	23	22	23	
,,	24	24	24	25	

Note.—On 23, viii, the pulmonary second sound seemed loud and slapping, but could not be heard more than half an inch away from the chest wall. On 29, ix, the loudness of the sounds last noted was probably in part due to nervousness produced by a prolonged examination.

TABLE IV.

Measurements of the cardiac sounds in compensated mitral regurgitation.

	Date.	Pulmonary area.		Aortic area.	
		2nd sound.	1st sound.	2nd sound.	1st sound.
Case 2, F., æt. 15	3, viii	12, 16, 24	8, 14, 14	9	8
	5, viii	14, 20	8, 10	14	2
	10, viii	16, 16, 18	10, 10	18	6
	4, ix	18 (20)	28	10	14
Case 3, M. . .	6, viii	16, 14	8, 10	14	8, 12, 14
	7, viii	12	10	10	8
	10, viii	18, 18	10, 12, 12	10, 12	14, 16
	14, viii	18 [22]	9	10	15
Case 4, M., æt. 26	8, viii	4, 6	4, 6	6	6
	29, viii	10	13	4	7
Case 5, F., æt. 33	22, viii	16, 14*	12	22	16
	5, ix	20 [28]	18	22	14
	19, ix	15, 15	24	21	14
Case 6, F. . .	23, viii	17, 18	8	10	4
	26, viii	14 (20)	12	8	8

* At this date the pulmonary second sound seemed much louder and sharper than the aortic, but on testing it with the apparatus used it was found that the aortic second sound had more carrying power than the pulmonary.

Table V.

Cases of mitral stenosis illustrative of the lessening of the loudness of the first sound as compensation improves, and *vice versâ*.

	Date.	Pulmonary area.		Aortic area.		
		2nd sound.	1st sound.	2nd sound.	1st sound.	
Case 7, F., æt. 31	8, viii	16	{1½ feet away}	14	48+	Very breathless.
	10, viii	16	24 (48)	20	—	Improving.
	23, viii	17, 18	48 + {⅛—¼ in.}	10, 12	32	,,
	27, viii	20 (22)	40 (48), {1/16 in.}	22 (24) (32)	40	,,
	30, viii	14	36	14 (16)	40	Got up on 30, viii.
	4, ix	24 (28) [32]	{2 feet away}	24, 28	48+	"Much upset."
Case 8, F., very severe case	23, viii	22	70 ± {⅛ in.}	8	24 to 28	
	25, viii	24 (20) [32]	50 + {¼—½ in.}	8 (12)	30	
	27, viii	22 (24)	32 {1/16—⅛ in.}	14	36	
	4, ix	14 (16)	28 (30)	8	28	Better.
Case 9, F., æt. 33, very severe case	29, viii	14	26	10 [12]	20 (22)	Breathless.
	31, x	20	18, 20	8	12, 16	Much better.
Out-patient, compensation fair.	5, xi	20 (22)	24	16	23	Pulse 100; rather breathless after walking.
	5, xi	24	22 (24)	22 (23)	16 (22)	Pulse 80; better again.

Figures in brackets { } refer to observations made without the use of the rubber bag.
See also Table VI, Case 10, last few observations.

Table VI.

Table showing the loudness of the heart-sounds in compensated mitral stenosis.

	Date.	Pulmonary area.		Aortic area.	
		2nd sound.	1st sound.	2nd sound.	1st sound.
Case 11, mitral stenosis and double aortic murmur	11, viii	16 (20)	20 [26]	8	14 [18]
	26, viii	12 (14)	18 [20]	8	12 [18]
Case 12, F., æt. 42	15, viii	20, 22	16, 22	24, 25	20
	22, viii	14 [16]	16, 22	20	13
Case 13, M., æt. 33, a severe case	24, ix	8 (12)	17	16	17, very well.
	7, xi	19	22	12	24, breath bad.
Case 14, M., æt. 40	19, ix	16, 16 (22)	23	18 (22)	26
	31, x	16, 16	18	16 (18)	21
Case 10, F., mitral and aortic	3, viii	8, 12	20, 22	8	10
	5, viii	10, 12	22	0 (murmur)	10
Compensation failing again	10, viii	24 (32)	32, 28 (24)	0 (murmur)	Murmur.
	14, viii	22 (28)	44	—	—
	4, ix	12 (14)	48 ($\frac{1}{10}$ in.)	—	—

XI.—*The Radical Treatment of Chronic Intestinal Tuberculosis, with suggestions for treatment in more Acute Disease and in Tubercular Peritonitis.* By A. W. Mayo Robson. *Read January* 10, 1902.

THE following cases of chronic tubercular disease in the intestine, treated surgically, have raised hopes in my mind as to the possibility of doing good, not only in chronic cases such as I am relating, but in some more acute cases, where, if the disease be strictly limited, a radical operation might remove it, but where, if more extensive, a short-circuiting might set the part at rest, relieve it from irritation, and so enable the ulcers to heal, the surgical measures being supplemented by general, medical, and hygienic treatment.

Whether these hopes will be realised or not, I trust that the cases in themselves may prove of sufficient interest to warrant me in bringing them before your notice. I think my hearers will agree that whatever may be their views on the treatment of acute intestinal ulceration, in chronic intestinal tuberculosis, especially if associated with stricture, surgical treatment is advisable.

Case 1.—*Tubercular disease of the appendix extending to cæcum and ileum; stricture of ileo-cæcal valve; removal of cæcum and adjoining part of ileum and ascending colon.*—Mr. G., æt. 23, was sent to me by Dr. R., county Durham, on June 19, 1900, with the history that after symptoms pointing to chronic appendicitis, an operation had been undertaken by an experienced surgeon, who found tubercular disease of the appendix without any general affection of the peritoneum or glands; but as there were numerous adhesions and some matting of viscera in the neighbourhood the appendix was not removed, and the abdomen was closed. The patient was in no way relieved, and steadily grew worse, generally and locally.

When I saw him he was very thin, and gave the history of having had pains in the cæcal region for eighteen months, associated at times with paroxysmal pain extending across the

abdomen, the paroxysmal attacks having recently become very frequent. There had been great loss of flesh, with night sweats and diarrhœa. A hard swelling could be felt in the cæcal region, and a coil of intestine below the umbilicus could be felt to contract and harden under the examining hand, indicating chronic obstruction.

Operation (July 17, 1900).—Oblique incision along outer border of right rectus; miliary tubercle on peritoneum with slight serous effusion, considerable matting of viscera adjacent to cæcum, tumour consisting of cæcum, lower third of ascending colon, appendix, and several inches of ileum removed, and open end of ileum connected to ascending colon by means of continuous sutures applied over a decalcified bone bobbin.

On account of adhesions it was difficult to explore the rest of the abdomen, but no other mass could be discovered, and the mesenteric glands, except those in the vicinity of the lower end of the ileum, were apparently not involved.

The wound healed by first intention, and the sutures were removed on the seventh day, but on the tenth day a small purulent collection was evacuated from the lower end of the scar, and the length of time that the slight sinus took to heal suggested tubercular infection. He returned home on August 29, markedly improved in his general health. Fresh air and general tonic treatment were advised.

On June 3, 1901, nearly a year later, the medical superintendent of a Durham sanatorium wrote to me to say that Mr. G. was improving generally, but that he had had some abdominal pain suggestive of obstruction by a constricting band.

A further exploration to ascertain if there are any bands or other removable cause of obstruction has not yet been pressed, as it has not appeared to be absolutely necessary, and the general health has improved.

The mass removed showed tubercular ulceration with great thickening of the cæcum and ileum, the disease having apparently started in the appendix and then extended to the cæcum and involved the ileo-cæcal valve, which would barely admit the tip of the little finger. I submit a photograph of the specimen.

Case 2.—*Multiple tubercular strictures of ileum; enterectomy.*—For the history of the following cases I am indebted to my dresser, Mr. J. Acomb.

Miss H., æt. 51, seen with Dr. Petch, of York.

Family history on father's side negative, on mother's side decidedly tuberculous.

Previous history.—She appeared to overgrow her strength between the ages of ten and fourteen years. When ten years old she had a glandular swelling in the neck, which was incised. No cough until twelve or thirteen years ago, since which time she has been troubled by bronchial catarrh every winter. No perspiration at night. Has recently lost flesh and strength very considerably. For some fifteen to twenty years the patient has suffered from so-called "bilious" attacks, accompanied by vomiting and pain across the abdomen, which would pass away after a few hours with diarrhœa. She had a bad colour at the time of the attacks, but had no apparent jaundice. These attacks had occurred for some years with perhaps a month's interval between each. Five years ago the character of the attacks appeared to gradually change, and three years ago their place was taken by a constant pain across the waist and back. The pain did not radiate, and was accompanied by indigestion and flatulence. She was subject to attacks of constipation for two or three days, followed by three or four days' diarrhœa, which would be accompanied by griping pain, and would come on quite suddenly and unexpectedly. No distension noted, no tenderness during intervals of pain, no tenesmus, stools as a rule watery, slight bleeding from bowel on defæcation five or six years ago, but no blood noticed since.

When seen by me on July 22 the patient was very thin, and it was easy to see vermicular contraction of the intestines, the tumour coming and going at varying intervals, the pain being at times very severe. On examining by the rectum, a nodular growth could be felt in Douglas's pouch, which was quite movable from side to side, and could be pushed out of reach.

Operation was advised, and was performed in York on July 26.

The abdomen was opened in the middle line, and the tumour previously felt by the rectum was discovered to be a coil of intestine, very much thickened and forming a mass as shown in the photograph, there being no less than four distinct strictures, the first within a foot and a half of the last, and all in the ileum. The affected area was brought through the incision, surrounded by sterilised gauze, and excised, the healthy bowel being sutured end to end over a decalcified bone bobbin.

I have not seen the patient since, but have heard that she recovered from her operation and gained flesh and strength, and is now well and strong.

The specimen removed was examined by Dr. Norman Smith, pathologist to the Leeds Infirmary, who gave the following report:

"There is a great increase of small round leucocytes and of connective tissue in the submucous layer. At places the leucocytes form dense, deeply stained aggregations. Seen by the higher powers, they are made up of a peripheral part composed of small cells, and a central portion of large cells. They are apparently giant-cell arrangements. At places in the muscular coat the same arrangement is commencing to be formed."

Diagnosis.—Chronic tuberculosis of the bowel.

The specimen is an extremely interesting one, as showing how near to stenosis it is possible to get without complete obstruction; it shows a gall-stone entangled in a pouch between two of the strictures.

Case 3.—*Multiple tubercular strictures of ileum; short-circuiting.*—On March 6 I saw Miss H. A., æt. 20, with Dr. Ernest Colby, of Malton. She gave the history that she had always been weak and thin since the age of ten, but this was accounted for by her having grown so rapidly. At the age of fifteen and a half she began to have pain on the right of the abdomen, associated with diarrhœa two or three times a day. From that time onward she had never been long free from pain and diarrhœa, and lately her attacks were coming on with greater frequency. Her loss of flesh and weakness were very marked, and she suffered from night perspirations. No tumour had been discovered, and she had not been troubled with vomiting. The motions contained neither blood nor slime, but were, as a rule, quite fluid like water. The pain came on directly after food for five minutes, then passed away, and two hours later diarrhœa usually followed. On examining the abdomen there was a rigid right rectus with tenderness in the cæcal region, but no swelling could be felt. Stricture was diagnosed and operation advised.

Although she tried fully various forms of medical treatment, with careful dieting and plenty of fresh air, there was no improvement, and on June 10 I operated on her at a surgical home in Leeds. The ileum was found to be strictured in several places, and the bowel generally thickened over a

distance of two feet before its termination in the cæcum. Miliary tubercles were scattered over the affected bowel and the adjoining mesentery, but the abdomen was not generally invaded by tubercle. There was some free fluid in the peritoneal cavity. As the patient was so very feeble it was thought she would not survive the removal of so much as two feet of the bowel; short-circuiting was therefore performed, the ileum being connected to the transverse colon by means of a decalcified bone bobbin. The wound healed by first intention, and the patient was sent home to the country on July 4, where a very good report has been received of her progress. She is now said to be well.

It was intended that she should return in order to have the diseased bowel excised, but her gain in health and the relief from the diarrhœa have made her shy of facing any further operation, which I think may not be necessary.

Case 4.—*Stricture of duodenum, probably tubercular, associated with tubercle of stomach and stenosis of pylorus.*—The patient, a feeble girl æt. 19, was seen in 1895. She only weighed 4 st. 10 lbs., and gave a history of vomiting and pain after food for four years, though the loss of flesh had only been very pronounced during six months. She was deathly pale, and so thin that the skin was tightly stretched over the bones. Tubercular glands were felt in the neck. The abdomen was swollen and the stomach was markedly dilated. On opening the abdomen the pylorus was found thickened and covered with tubercular nodules, which were scattered over the omentum and other viscera. Some free fluid was found in the peritoneal cavity. On laying open the pylorus to perform pyloroplasty, the finger was passed into the duodenum and encountered a stricture one inch beyond the pyloric ring; this was evidently the result of ulceration. The tip of the little finger could just pass through it. A Weiss's dilator was employed to stretch the stricture, which was dilated so as to allow the thumb to readily pass through it. The pyloric incision was then closed by sutures transversely to the axis of the canal. For a week the after-operative progress was satisfactory, the wound healed, and the stitches were removed, but no strength was gained, and she gradually failed and died from exhaustion in the second week.

With my present experience I should have performed gastro-enterostomy and begun feeding at once, and I believe the result would have been different.

CASE 5.—*Tuberculosis of cæcum; enterectomy.* (Report furnished by Mr. J. F. Dobson, F.R.C.S., Resident Surgical Officer).—H. S., æt. 22, a dressmaker, was admitted to the Leeds Infirmary November 15, 1897. The patient had suffered from dyspepsia for several years. About six months ago she noticed that her side was painful when she turned, and that she could not bear the pressure of her stays. This became worse, and was accompanied by attacks of rather acute abdominal pain, principally in the right side. She never vomited during the attacks, and had never been jaundiced. The pain had no relation to meals. Bowels regular, generally rather loose; no blood or mucus noticed; appetite poor and considerable wasting of late. Menstruation commenced at seventeen, lately scanty and painful. Tumour felt in region of cæcum, but tenderness not marked. At the operation the tumour was found to involve the cæcum and commencement of ascending colon. The lumen of the gut was not occluded but narrowed. No general tuberculosis of peritoneum observed, and no enlargement of glands was found. The tumour was excised, and the ileum and ascending colon united by continuous sutures over a decalcified bone bobbin. The wound healed kindly, and the patient improved rapidly, and was discharged in fair health on January 8. Although the health was regained, and there was no further return of abdominal trouble, the patient had pleurisy some months afterwards, and is said to have died nearly a year later at her home.

Pathologist's report.—A portion of tissue removed from wall of cæcum is of a pale grey colour, fairly elastic and translucent.

Microscope.—Delicate fibrous tissue with a few round- and spindle-cells between. Many rounded and oval areas occur where collections of round-cells are tightly packed. In the midst of each group one or more well-developed giant-cells are found.

Appended is a photograph of the specimen removed at the operation.

CASE 6.—*Tubercular disease of rectum and sigmoid flexure of colon; colotomy and partial colectomy.*—F. O., æt. 37, was admitted to the Leeds Infirmary on October 31, 1898, suffering from a profuse purulent discharge from the anus associated with great pain, increased on defæcation, and with diarrhœa and great deterioration of health.

He gave the history of having had an operation for "fistula in ano" three years before, from which he recovered, but six months later he noticed the motions to be streaked with blood and slime, which gradually increased in quantity until it began to come independently of normal bowel action.

On examination by means of a speculum, the whole of the rectum was found to be involved in ulceration, which extended upwards beyond reach of the proctoscope. The sigmoid flexure could be felt to be much thickened, and was tender to pressure.

Inguinal colotomy was performed, the sigmoid being drawn out as far as possible in order to secure healthy bowel being opened; the diseased part external to the abdominal wall was then cut away. This was examined by the resident pathologist and proved to be tubercular. After the first week the lower bowel was regularly syringed out with boric acid solution containing a tablespoonful of tincture of hamamelis to the pint, the stream being forced alternately from anus to colotomy opening and *vice versâ*.

Convalescence was very slow, but he steadily picked up strength, and after going to the convalescent home was discharged much relieved.

Remarks.—Apart from cancer, stricture of the bowel usually results from ulceration, with consecutive changes in the coats of the bowel, but there is a specimen in the Leeds School Museum which shows a stricture due to gradual invasion of the bowel walls by tubercle spreading from glands in the mesentery, and I have also seen stenosis produced by fibrous hyperplasia extending from a mesenteric tubercular gland in process of cure.

In the first four cases here related there was no serious difficulty in coming to the conclusion that stricture was present, and, from the length of time that the obstructive symptoms—constipation alternating with diarrhœa and pain—had existed, that the narrowing was simple in character and not malignant. In three of the cases, the family and personal history, the early age, the loss of flesh, and night perspirations were suggestive of the stricture being tuberculous; moreover there was an absence of history of typhoid fever or dysentery, the two other chief causes of intestinal ulceration, and of hernia or injury, which sometimes lead to intestinal stenosis. In the fifth case it was suspected that the tumour might be malignant, and, in fact, it was only on careful

examination subsequently to removal that its true character was discovered.

The cases related afford examples of tuberculous ulceration in parts of the intestinal tract from the duodenum to the anus, though the most common site of the disease is in the lower ileum and about the ileo-cæcal valve.

Chronic tuberculous ulceration leading to stricture is evidently not very common, though perhaps some cases put down as malignant, but where cure occurs after colotomy or enterectomy, may be of this nature. I have in my mind two cases of apparent cure of malignant stricture by colotomy, one in a man of 33, where I subsequently closed the colotomy opening after the health of the patient had become fully re-established, and when there was manifestly no further need for the artificial anus; and another in a man of 58, where the tumour exposed at the time of the colotomy has apparently disappeared and the patient is anxious to have the artificial anus closed.

Now although both of these cases had all the appearances and feel of carcinoma, I think the subsequent course of events makes it extremely probable that the intestinal thickening leading to stenosis was tuberculous, though proof is wanting. The extension of the tubercular process from the appendix in Case 1 is apparently not a common condition, though seeing that the appendix is composed largely of lymphoid tissue, it makes one wonder that it should comparatively rarely be attacked by tubercle, especially when the inflammatory attack is repeated over and over again. I have, however, operated on at least five cases of tubercular appendicitis, and the removal of the diseased part has issued in recovery and gain of health. The following is a good example:

Case 7.—*Tubercular appendicitis; appendicectomy.*—A youth æt. 17 was sent to me on October 10, 1901, from the West Coast in such a weak condition that it was feared he could hardly bear any operation; he was suffering from night sweats and had a hectic temperature. There was well-marked tenderness over the cæcum, with rigidity of the muscles. The history given was that he had had acute appendicitis eighteen months before, and that the abscess had been drained but the appendix had not been removed. A recurrence of the attacks with deterioration of health led to two attempts at removal of the cause, but the appendix, it was said, could not be found. At the operation on October 6 there was well-

marked tuberculosis of the cæcum, with numerous miliary tubercles scattered in the neighbourhood, though the rest of the peritoneum was free. After carefully detaching numerous and firm adhesions, the appendix, which was tubercular, was found in the centre of offensive thick caseous pus, behind the cæcum and ascending colon. After removal of the appendix, clearing out and purifying the pus-containing cavity, the abdomen was closed, a single gauze drain being used. Recovery was uninterrupted, and he returned home at the month's end. A report was received from Dr. Vinter in December to say that the patient was well, and getting out as usual.

The stricture in Case 1 was caused by a stenosis of the ileo-cæcal valve, but the lumen of the adjacent ileum and cæcum were also seriously narrowed by thickening of their coats.

Cases 2 and 3 are characteristic examples of tubercular ulcers, which usually begin in the lymphoid follicles and adopt a transverse direction, following the course of the vessels around the whole circumference of the bowel, thus leading to stricture in chronic cases, from the lymph, which is thrown out to limit the ulcerative process, tending to contract.

The situation, in the ileum, and the multiple characters of the strictures were also characteristic of tubercle.

While in Cases 1 and 7 the adhesions to adjoining parts were most intimate, in the other cases the affected part of the bowel was quite free, thus rendering operative measures quite easy and simple.

In Cases 1, 2, 3, and 5 my decalcified bone bobbin was employed as a temporary splint over which to apply sutures, a method which I have described on numerous occasions, and which I still employ and advocate as being simple and efficient.

The physiological rest to the diseased parts secured by short-circuiting, as in Case 3, and by colotomy in Case 6, seemed to answer so well that where the radical operation of excision would involve serious difficulties, or the patient is not in a condition to bear it, I would not hesitate in future to be content with similar procedures, and then to trust to general treatment and hygienic surroundings to assist in the cure.

If in the future operative treatment be thought advisable in acute cases, I anticipate that short-circuiting the diseased

DESCRIPTION OF PLATE VII.

To illustrate Mr. Mayo Robson's paper on the Radical Treatment of Chronic Intestinal Tuberculosis.

Fig. 1.

Fig. 3.

Fig. 2.

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area will be found to be the more useful procedure, as it is the easier and safer method to pursue; but experience only can show whether my suggestions will prove of so much service in acute as they are in the chronic cases.

It is interesting to note that in Cases 1 and 7, where there were tubercular nodules in the peritoneum, where, in fact, there was tubercular peritonitis co-existent with the bowel disease, the mere exploratory operation had not cured or even arrested the tubercular process, but that the radical operation of removing the principal focus of disease, or, as in Case 3, the short-circuiting, and so setting at rest the diseased bowel, was in all three cases effectual in putting a stop to the tubercular peritonitis, and, so far as one can say without seeing the parts exposed, in curing the more diffuse disease.

Now all surgeons must have had the experience of curing tubercular peritonitis by simple abdominal section with or without drainage, and probably many of us have also experienced disappointment in other cases where the effusion has returned after a longer or shorter interval. May the explanation of these recurrent cases lie in the fact that the original focus of disease in the ovaries, Fallopian tubes, or bowel has not been removed, and has been again the starting-point for a further diffusion of the tubercle throughout the peritoneal cavity?

If this be a correct explanation the remedy is quite clear: that wherever the original disease may be it should be removed, if that be possible, without adding seriously to the risk of operation.

XII.—*A case of Hair-ball removed from the Stomach of a child æt. 9 years.* By E. Percy Paton, M.S. *Read January* 10, 1902.

CASES of hair-ball being very infrequently seen in the human stomach, I have thought it might be worth while bringing before the Society the following case.

I was asked to see J. T., a very intelligent girl æt. 9, on account of a mass in the abdomen, which my friend Dr. W. R. Brunton, of East Twickenham, under whose care she was, thought to be not improbably a hair-ball in the stomach.

The mass had been found accidentally while examining the child with a view to her going to a school.

Her mother gave the following history:—At the age of three it was noticed that the child had acquired a habit of eating hair (either her own or other people's), string, "fluff" off mats, etc. This continued until she was five, when all her hair was cut off in order to break the habit, since which time the mother believed that she had given it up entirely. Since the child was four years old she had had occasional attacks of pain shortly after food, and had been treated by various doctors for indigestion; the lump had been noticed, but was thought to be undigested food; her only other trouble had been occasional slight so-called feverish attacks of a very mild type.

On palpating the abdomen a mass was felt lying just above the umbilicus, and extending across the epigastric region and into the right and left hypochondriac regions. It was very firm in consistency and fairly smooth in outline, being rather more than three fingers' breadth in width towards the left, and a little less to the right. It was very freely movable from side to side, but could not be dragged down below the umbilicus. When pushed to the left it became partly concealed under the ribs, but on moving it to the right that end of the swelling passed somewhat downwards and into the loin. There was no pain on manipulating the mass.

On October 9, 1901, a median incision was made through the linea alba above the umbilicus, and the stomach was at

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DESCRIPTION OF PLATE VIII.

To illustrate Mr. Paton's case of Hair Ball removed from the Stomach of a Child æt. 9 years.

once felt to contain the mass; it was therefore opened in its long axis by a three-inch incision and the mass shown extracted; the gastric wall was then closed by a continuous suture for the mucous coat, and interrupted Lembert sutures for the muscular and peritoneal coats, and the abdominal wall closed in layers.

The child was somewhat collapsed after the operation, but soon rallied, and made an uninterrupted recovery, being fed by the rectum for the first two days, except for hot water by the mouth, and then by increasing quantities of fluid and solid food by mouth. I do not, however, think there would have been any great objection to giving food by the mouth almost from the first if it had been necessary.

The specimen, on examination, was found to be practically odourless, and to consist largely of the child's own hair, but also of a considerable admixture of what appears to be horse-hair, probably obtained from the stuffing of chairs. It weighed 7¼ oz., and measured 9 inches along its greatest curve, and 6 inches round its thickest part.

The depraved taste which led to this formation is very curious in a child distinctly above the average in general intelligence. When asked after the operation why she had been in the habit of eating hair she said, "Because it was so nice."

XIII.—*A Note on the Results of Treatment in Seventy-four Cases of Appendicitis with Localised Suppuration, with special reference to the question of recurrence if the appendix is not removed.* By GILBERT BARLING, M.B. *Read February* 14, 1902.

DURING the last few years I have operated upon upwards of 140 cases of appendicitis in the acute stage, and it has appeared to me that some information as to the immediate results of the operations, the complications met with, and the subsequent fates of the patients would be of interest.

The task of collecting the notes of all these patients, and of following the cases subsequently, became so considerable that I found it necessary to limit the field of my observations, and I propose only to deal to-night with those cases in which a well-localised condition of inflammatory mischief has been found; practically there has been in all of them an abscess localised to the right iliac fossa or to the parts immediately adjacent. I purposely exclude all cases of acute perforation or gangrene with wide-spread septic peritonitis, and those also in which the infective condition, though not generalised, yet had spread widely into the pelvis and had no well-defined limits. Cases such as these last cannot be grouped together with localised abscesses if we are to form conclusions of any value on this serious and common disease.

This reduces the cases for consideration to seventy-four, and it is necessary to subdivide these again into two groups:

(*a*) Comprising forty-six cases of localised abscess well shut off by adhesions, but not yet come forward so as to be adherent to the parietal peritoneum, so that operation involved the opening of the general peritoneal cavity. These I refer to later as *non-adherent abscesses.*

(*b*) Comprising twenty-eight cases of localised abscesses which had become adherent to the parietal peritoneum, and in which the pus was evacuated without opening the general cavity. These I refer to as *adherent or safe abscesses.*

The points to which I particularly wish to direct attention to-night are—

1. The time at which abscesses should be evacuated.

2. The safety with which this can be done if removal of the vermiform appendix is not insisted upon.

3. The rarity with which recurrence takes place if the vermiform appendix be not removed.

4. The complications at the time of or following operation.

5. Certain risks to life which follow attacks of appendicitis, apart from recurrence.

The time at which abscesses should be evacuated is, in my opinion, as soon as a diagnosis of suppuration has been formed. The diagnosis as a rule is not difficult; the initial symptoms of pain, local tenderness, and vomiting are generally well marked; the temperature, at first raised sharply and continuously, soon takes the hectic type. The pain and tenderness remain acute, or if they be so lessened as to allow of palpation through the now less rigid muscles, a mass of some size can be palpated in the right iliac fossa, on the margin of the pelvis, or possibly in its cavity, and there is a tendency for the swelling to increase rather than to subside, as it would do if resolution were in progress. The pulse may remain quick, but a fall in its rate to quite normal is not inconsistent with localised suppuration. A marked leucocytosis is a valuable confirmatory sign, especially if there is opportunity to make more than one observation and the leucocyte count is found to be rising. In only two out of twenty-one cases have I found a normal leucocyte count associated with suppuration. In one of these cases only a single observation was made; the other indications of suppuration were fairly clear and were acted upon, pus being found. In the other case, a male æt. 20, on the seventh day of the disease operation was resorted to for suppuration, the diagnosis of pus being based on the persistent rigidity of the belly and a dry tongue, although the temperature had not been above normal, nor had the pulse been above 80 for three days. The leucocyte count on three consecutive days was not on any day as high as 8000, and yet 3 to 4 oz. of pus was found. In three of my cases operated on for recurrence, when only a catarrhal and interstitial appendicitis without suppuration existed, repeated observations failed to show leucocytosis. I am therefore inclined to lay considerable stress on the value of leucocytosis as indicating appendical suppuration, but it is not pathognomonic, and the condition of the leucocyte count is only one factor in the diagnosis, to be given its due weight and not more. I ought to add this much further, that when the leucocyte count has been raised pus has always been

found; in this, therefore, as in many other conditions, positive evidence is of more value than negative.

Whilst it is generally true that a considerably raised temperature at night with a morning remission is present in these suppurations, it must be acknowledged that the temperature may be so nearly normal that it is of no assistance. Close scrutiny of the temperature chart is, however, necessary, because in a few cases it may be found that, whilst the temperature does not rise quite to 99 in the evening, yet in the morning the record may be as low as 97 or near that, thus giving a remission of one and a half to two degrees. Should this continue for two or three days it is very suggestive of quiet or latent suppuration, a condition which is apt to be overlooked, especially in old or feeble patients, until a large abscess has formed. If the patient looks ill, is anæmic and pasty-faced, this also is suggestive of suppuration, even with a normal temperature. Rigors are exceptional, fluctuation, redness, and œdema are late symptoms, and should not be waited for.

Early operation in localised suppuration is called for, first because of the danger of rupture of the localised collection into the general cavity, a condition which is the most fatal of all the accidents of appendicitis. There is, further, the extension in various directions, with progressive destruction of tissues, bringing increased risk to life and tedious convalescence. These extensions may be into the pelvis, the right hypochondrium, into the lumbar space, and then over the crest of the ilium to the lumbar and gluteal regions. On two occasions I have been called to cases in which the abscess has found its way through the anterior abdominal wall into the subcutaneous tissue, and there given rise to the most extensive gangrene and emphysema of the tissues of the abdomen, thorax, and lower extremities with a fatal result in each case. One of these patients, a boy æt. 15, had only been ill eight days, and had not received any medical treatment until the gangrenous process developed.

It may be suggested that less risk is run if intervention is delayed until the abscess has come forward and become adherent or safe. I hope to be able to show, however, that, with proper precautions, abscesses may be approached across the open peritoneal cavity with great safety, so safely, indeed, that delay is not justifiable.

I turn now to the second point raised: the safety with which appendical abscesses may be treated if removal of the

appendix is not insisted upon. My own plan is to incise directly on the most prominent part of the swelling present, making an incision of moderate extent, to be enlarged if it seem advisable, and in these acute cases I rarely attempt the mere separation of the fibres of the internal oblique and transversalis, which may prove embarrassing from the narrow space provided for manipulation, but prefer a vertical division of all the layers of the abdominal wall.

If the appendix can be recognised easily I remove it, but I am chary of prolonged search for it, especially in adherent abscess, when the incision does not involve the opening of the general cavity.

When dealing with a non-adherent abscess, in which, of course, the general cavity must be opened, I allow myself greater latitude, but even then I do not pursue the search far. Fæcal concretions in the abscess cavity I am always careful to search for and remove.

I suture the incision as carefully as the condition of the parts permits, and I practically always drain, using a rubber tube rather than packing for the purpose.

Of adherent or safe abscesses twenty-eight have been treated, with one death. Of these cases only in three was the appendix found and removed, though in a considerable number concretions were discovered.

Of non-adherent abscesses forty-six have been treated, with one death. In these the appendix was removed twenty-two times, and in several others concretions were found lying in the abscess cavity. To sum up, of the seventy-four cases operated on two died.

Of the two deaths just referred to, one in each group, one patient was an old and feeble man in whom a large abscess formed with very little to suggest its presence, and the patient died two days after, apparently from asthenia, but no autopsy was allowed. The other patient died apparently of septicæmia on the fourth day after operation, and it may be that he had peritonitis spreading from the operation area, but no autopsy was held. It may therefore be asserted confidently that so far as the immediate result of operation is concerned there is little to be urged against this conservative method. If removal of the appendix is insisted upon, there is more risk of infection of the general peritoneal cavity from the more extensive manipulations required, which are likely to break down limiting adhesions; the walls of inflamed and adherent intestine are easily torn, with resulting fæcal fistula;

and the constant oozing of blood, which in some cases is really quite embarrassing, makes the search for the appendix a tedious and severe operation. It must be acknowledged that when the appendix is not removed convalescence is apt to be more tardy, and in a few cases a sinus remains for a more or less prolonged period; but better this than added risk to life, and the appendix may be removed, if necessary, when the parts have settled down after the acute symptoms have entirely subsided.

The next point is the proportion of cases in which relapse is likely to occur if the appendix is not removed. The exact percentage I do not pretend to give, but I have sufficient evidence to show that relapse is quite rare. The patients in my lists in whom the appendix was not removed number forty-nine; of these thirty-three were hospital cases and sixteen were private patients. Any one who has attempted to follow hospital cases some years after they have left the institution will realise how difficult it is to give the details of the first group. It is, however, satisfactory to find that of these thirty-three hospital patients all but two have been traced, and of these I can learn nothing, not even whether they are alive or dead. Of the hospital patients traced, none have suffered from recurrence of the disease, though one, an old man, had died of bronchitis and other troubles, and one other had died of strangulated hernia, and reference will be made to his case later on.

All the private patients have been traced, and of these one suffered from relapse. This patient, a girl with an adherent abscess, had acute recurrence within a few weeks of her first operation, and I then again operated, finding a fairly wide-spread peritonitis with a small purulent collection around the appendix, which was then removed and recovery followed. The time which has elapsed since operation on these forty-nine patients varies from ten years to a few weeks; therefore relapse may yet take place in some of them, but I think sufficient has been said to show that such an event is rare.

The patients who cannot be traced may have had recurrences, but with the history of the others before us it would not be reasonable to assume this, and probably it will be best to ignore them. That being so, we have in forty-seven cases only one case of relapse.

Complications at the time of operation or following it have not been very numerous. In one patient, a male

æt. 38, a considerable amount of albumen was found in the urine, but this entirely cleared up in a few days. In one patient, a girl, the abscess had opened into the bladder, but recovery followed, though somewhat tediously, on drainage of the abscess cavity, the appendix not being removed. In two patients secondary suppuration occurred in the right hypochondrium a few days after the opening of adherent abscesses, the appendix not being removed, but recovery followed the evacuation of these further collections. Phlebitis of the veins of the lower extremities and pleurisy with pneumonia have been the commonest complications, each occurring three times, and in two patients phlebitis and lung trouble coincided, but all the patients recovered without suppuration in the pleura or vein. Of all the cases operated on only one has a fæcal fistula, and in this patient I persisted in removing the appendix, which ran upwards behind the ascending colon, and in doing so perforated the bowel with my fingers. An attempt to close the opening by an extra-peritoneal operation failed, and the man at present declines further intervention.

Inquiry into the fate of patients who recovered from operation shows, as was to be expected, that a distinct mortality threatens them as a direct sequela of the disease. One patient, a woman æt. 32, was under my care four years after operation for non-adherent abscess, the appendix being removed, with symptoms of internal strangulation. She had been acutely ill some days, and was about four months pregnant. Operation found a small band of adhesion arising from the cæcum, which had kinked and produced a partial volvulus of a portion of ileum, and the intestine was gangrenous. Resection with immediate union was performed, but the patient aborted on the third day and died on the fourth day after operation. Another patient, a boy æt. 15, operated on for a non-adherent abscess, the appendix being removed, made a good recovery. A few weeks later he was readmitted to hospital with vomiting and great distension. Starvation and repeated enemata relieved his symptoms, and he was discharged with a warning against over-feeding and constipation. Both these directions were ignored, and in a short time the patient was readmitted with his old symptoms in an intense degree. He was so ill that immediate operation was called for. Dense and wide-spread matting of small intestine was found in the right iliac region. The only possible method of relief was by a lateral anastomosis after evacuation

of the intestine, but the patient died the next day. A third fatality followed four years after operation in a middle-aged man, who was very ill at the time of his operation, and in whom a ventral hernia developed through the scar. He neglected to wear a support, and came under the care of one of my colleagues with strangulation in the hernia, and died after operation for its relief.

XIV.—*The Treatment of Intra-peritoneal Abscess of the Appendix, illustrated by cases.* By F. C. Wallis. *Read February* 14, 1902.

IN the accompanying small series of consecutive cases of abscess of the appendix it will be noticed that in every case except the last the abscess was intra-peritoneal, by which is meant that the abscess which is formed in connection with the appendix, although shut off by surrounding adherent coils of intestine, was in no way adherent to the parietes, or only so slightly as to form no protection.

In every case, therefore, the general peritoneal cavity had to be opened before the abscess could be dealt with. The following are details which were performed in each case:

A comparatively small incision is made through the skin down to the rectus sheath, which is divided; the length of the initial incision depends mainly upon the thickness of the abdominal parietes. The rectus muscle being pulled inwards, the posterior part of its sheath is opened, the peritoneum incised and held apart by forceps. If now the finger is introduced, and a search more or less roughly carried out for whatever may be found, the consequences will be disastrous: the abscess cavity will almost inevitably be opened, and the contained pus escape into the general peritoneal cavity. Great care is necessary to avoid this catastrophe, as *in the young*, even after many days, the adhesions are of the frailest character, and unless the intestines are manipulated with gentle hands these adhesions give way and expose the general peritoneal cavity to the infection of the escaping pus.

To avoid this, when the peritoneum has been incised, a blunt hook with a large curve is used to pull up the parietes on the left side of the incision. The general peritoneal cavity is shut off all round by introducing pieces of Gamgee tissue about 1½ to 2 inches wide above, internally, and below, thus forming a crescent around the affected area.

Now the abscess may be looked for by gently separating the adhesions in the appendix neighbourhood. This must be done *quite* gently, otherwise the pus, which is always under

a certain amount of tension, may escape too rapidly to control it. If this does occur, as it sometimes will in spite of all proper care, the patient should be immediately tilted over to the right side, and the pus directed to a small bowl held underneath.

When the pus escapes gently it can be sponged up as it wells out of the cavity until the tension has ceased, when the opening is separated more widely, and the cavity is thoroughly cleansed by sponging with Gamgee tissue (marine sponges should not be used).

If there is any appendix to be removed, this should be done now; the cavity is then dusted with iodoform and filled with iodoform gauze.

In one or two cases the cavity was swabbed out with pure carbolic acid, but the subsequent oozing was so excessive that I gave it up.

It is important, especially for the first twenty-four hours, to keep the surroundings as dry as possible, and iodoform powder with moderately light packing of the gauze answers well. This packing is removed on the fourth day, *i. e.* after the bowels have acted, as a rule.

In some of the cases peroxide of hydrogen was used to clean up the abscess cavity. I have used it now for some years, and can recommend it. The abdominal incision is closed by one set of interrupted fishing-gut sutures, care being taken that each layer of the parietes is picked up by the needle—a large curved Hagedorn—as each suture is passed.

It will be noticed that seven out of the eight patients were young—a matter, I believe, of some considerable clinical importance. It is not intended in this paper, which is only a record of one's clinical experience, to deal with the vast amount of literature on the subject. But from what I have read and from what I have gathered from the clinical experience of others—and it certainly is my own,—suppurative appendicitis is much more common in young people than the recurrent form, and hitherto sufficient importance has not been attached to this fact.

In all these cases the attack which ended in suppuration was the primary one.

The onset in each case was a sharp, definite attack of abdominal pain, and in most cases this—perhaps naturally enough—had been looked upon as an ordinary stomach-ache by the patient or relatives, and the sufferer had continued to

go about for several days before applying for medical relief. Herein lies the danger. If by chance adhesions form—and fortunately this is the rule—before the pus escapes from the appendix a bulwark of safety has been made, which is of great value.

But many of us have had personal experience of those cases, when a young life is lost in two or three days by a suppurating appendix bursting before any sufficient adhesions were formed.

Another point which I have already mentioned is the *frail* character of the surrounding adhesions *in the young,* even after many days. Case 1 was operated on ten days, and Case 4 four days after the original attack. Yet in each case, as in all the others, save one, quite delicate manipulation was necessary to avoid bursting the adhesions before the necessary guards had been placed around the affected area. The importance of recognising this clinical fact is evident.

The one exception to this was Case 7. The age of the patient was 48, and here the adhesions were firm and strong, forming a good protection. In my experience this also is the rule in patients about this age.

These facts emphasise the necessity of operating quite early after the primary attack of appendicitis in young children.

In all these cases a thorough view of the abscess cavity was obtained, and whenever any appendix or portion of it remained this was removed at the time. When the pus tracked up behind the colon a posterior incision was made, from which the wound drained, and the anterior incision was closed.

The sequel of commencing intestinal obstruction, which occurred in Case 5, shows the advisability of removing any omentum which may, owing to its inflammatory condition, be capable of contracting adhesions with neighbouring coils of intestine. A certain amount was removed in this case, but, as the sequel shows, not enough. The rapid convalescence which ensued in all these cases was, I believe, due in great measure to the thorough cleansing of the abscess cavity at the operation, and to the removal of whatever was left of the appendix.

With regard to the somewhat vexed question as to whether the appendix should or should not be removed at the time when the abscess is opened, I do not think that any hard and fast rule can be made. In four of my cases the appendix

was seen when the abscess cavity was exposed, and was comparatively easily removed. But any systematic search when the appendix was not apparent was not made, and I do not think it would be good surgery to make one, as very serious harm might be done by opening up the peritoneal cavity, and recurrences after an abscess has formed when the appendix has not been removed are rare.

The treatment of the last case on the list is one which I have adopted lately when pus or puriform fluid was found in the general peritoneal cavity, but at the time there was no general peritonitis. My reason for such treatment is my great and growing respect for the wonderful protective and recuperative power of the peritoneum when not irritated by chemical solutions and rough manipulation, which in these cases does nothing but harm.

Cases illustrating Mr. F. C. Wallis's paper.

Case 1.—T. A., æt. 15, admitted to Charing Cross Hospital, July 6, 1900. Operation July 13. Discharged August 12.

Pain in abdomen on July 3, given castor oil. Sickness on July 4 and 5. Temperature varied from 101° at night to 98·6° in the morning. Transferred to surgical side on July 13.

Abdomen.—Somewhat distended; slight respiratory movement; markedly tender, especially over right iliac region, where the skin is slightly œdematous, and a distinct fulness can be made out.

Operation.—Skin incised for two inches over McBurney's point. Rectus sheath opened and muscle pulled inwards. Peritoneum opened, when thickened œdematous omentum presented itself. Incision enlarged 1½ inches. In the middline was a mass of omentum, underneath which was a gangrenous appendix and a small quantity of very offensive pus. The appendix was removed, the abscess cavity swabbed out, and a portion of thickened omentum was cut away. The cavity was dusted with iodoform powder, packed with iodoform gauze, and the greater part of the incision brought together with silkworm-gut sutures. Iodoform gauze removed three days after operation, and a small piece reinserted into the cavity for drainage purposes. The cavity filled rapidly, and patient was discharged on August 12.

Patient was operated on on January 25, 1901, for small ventral hernia.

CASE 2.—J. S., girl, æt. 13, admitted October 11, 1900. Operation October 16. Discharged November 13.

Suddenly taken with abdominal pain on October 2. Kept about until October 7, when she went to bed and diarrhœa commenced. Was admitted to Metropolitan Hospital as a case of enteric fever. When seen after admission, a definite swelling in the right iliac fossa was discovered, and appendicitis diagnosed.

Operation on October 16, when an abscess was found situated between the adherent small intestines and cæcum. This was treated in the manner described above. An uninterrupted recovery. (Peroxide of hydrogen was used as a lotion.)

CASE 3.—F. L., girl, æt. 15, admitted to Sutton Cottage Hospital on September 4, 1900. Operation September 9. Discharged October 9.

Ill with abdominal pains and vomiting on September 2; pain and resistance more marked in right iliac fossa. When seen on the 9th there was a well-marked swelling in right iliac fossa, and temperature 103·2°, but patient was lying on affected side reading a book.

Operation the same day, when an abscess was found containing nearly 1 oz. of characteristic pus. This was treated on the same lines as the others. The child made an uninterrupted recovery.

CASE 4.—A. B., youth, æt. 17, admitted February 6, 1900, to the Metropolitan Hospital. Operation February 23. Discharged April 3.

Seized with acute pain in lower abdomen during the night of January 30, and this was followed by vomiting. Pain and daily vomiting continued until admission, when an indefinite swelling was found in the right iliac fossa extending as far as the middle line. This swelling was dull on percussion.

Operation (February 23).—The omentum, which presented through the wound, was found adherent to the anterior abdominal wound, and also to a loop of small intestine; this loop was adherent to the lower part of the cæcum. Between these coils was a small abscess and the remains of the appendix. This was removed, and the cavity packed with gauze. Patient made a good recovery, but the sinus was longer than usual in filling up. (Peroxide of hydrogen was used as a lotion.)

Case 5.—J. Y., youth, æt. 17, admitted to Charing Cross Hospital on October 26, 1900. Operation October 26. Discharged January 9, 1901.

The previous history of this case was not clearly made out, but on admission the patient was quite ill, and had obvious signs of suppurating appendicitis.

At the operation the omentum, small intestines, and cæcum were all much matted together, and on separating these a considerable abscess cavity was exposed, filled with foul pus. In the cavity was the gangrenous appendix; this was removed, and the cavity swabbed out and packed with iodoform gauze. A portion of the sodden and thickened omentum was removed.

The patient did very well until the third week, when he had symptoms of intestinal obstruction, necessitating a laparotomy, which was performed in the mid-line, when two coils of intestine were found adherent to the great omentum; these were separated. The patient then made a good recovery.

Case 6.—G. S., girl, æt. 11, admitted to Metropolitan Hospital July 1, 1901. Operation July 2. Discharged August 4. First complained of pain in right iliac fossa on June 25, which gradually became worse until June 29.

There had been no sickness. Bowels had been opened twice a day. On day of admission had had lamb and mint sauce twice. At this time her temperature was 103°, pulse 132.

Abdomen has a distinct fulness and sense of resistance in right iliac and lumbar regions, where manipulation causes pain.

On opening the abdomen the colon presented itself. This was pulled towards the middle line, and an abscess cavity situated externally to the colon was found, which held 2 oz. of foul pus. The cavity extended up towards the liver, and was lined with a thick white membrane. A posterior incision, for better drainage, was made in the loin, and the abscess cavity treated in the same way as the other cases. Recovery uninterrupted.

Case 7.—A. W., female, æt. 48, admitted July 3. Operation July 4. Discharged August 14.

Sudden severe pain in lower abdomen on June 25, which settled in the right side; no sickness, but rather violent retching.

Abdomen moves freely on respiration; on palpation a

large mass can be felt in right iliac and lumbar regions, which extends upwards to lower surface of liver.

The abdomen was opened by an incision 4½ inches long. When the transversalis muscle was reached it was found to be much infiltrated and thickened; on opening the abscess cavity firm adhesions formed definite and safe boundaries (the only case of this series where this was so); abscess cavity treated in usual manner. Recovery uninterrupted.

Case 8.—W. S., male, æt. 20, admitted to Metropolitan Hospital November 5, 1901. Operation November 5 (same day). Discharged January 4, 1902.

Sudden attack of pain with shivering and nausea November 3, since when the bowels have not acted and no flatus has been passed. Vomiting commenced on November 5.

On admission looked very ill; temperature 97·5°, pulse 120. Abdomen distended, rigid, not moving with respiration. Very tender all over right iliac fossa.

Feet and hands peeling in a manner suggestive of scarlet fever. Was sent in notified as a case of typhoid fever.

Operation.—When the abdomen was opened the intestines in the right iliac fossa were found covered with thin puriform fluid. No signs of adhesion or appearance of localisation of peritonitis. A long swollen appendix was found lying on the outer side of the cæcum, between it and the parietal peritoneum; this was removed. A quantity of sero-purulent fluid was withdrawn by a syringe from the right side of the pelvis, and the wound treated as in the other cases, with the addition that a large drainage-tube was passed down into the Douglas's pouch. Was constantly sick until November 8, when, after repeated treatment, the bowels acted well. Recovery from now on was uninterrupted. When, on November 15, the stitches were removed, it was found that no union had taken place, the whole wound gaped, exposing the intestines. No ill-effects followed this unusual *contretemps*.

XV.—*Two cases of Arthritis of the Knee.* By H. W. Page, M.C.Çantab. *With an Investigation into the Pathogenesis.* By Alexander Paine, M.D., and F. J. Poynton, M.D. *Read March* 14, 1902.

From time to time during the past few years I have had cases of chronic synovitis of the knee-joint under my care in which the condition could be attributed neither to uncomplicated injury nor to rheumatism, osteo-arthritis, tuberculosis, or other every-day disease. The chief characteristic of the affection has been the absence of any tendency to subsidence of the synovial effusion under the influence of rest, warmth, the application of ice, blistering, and other local remedies, together with an unusual amount of pain. Furthermore, the state of the patient has not uncommonly been such as to suggest a subacute form of toxæmia, and when by aspiration the fluid has been removed it has usually been of such a kind—thick and greenish in colour, cloudy, with flakes of lymph and leucocytes—as to lead me to wash out the synovial cavity with carbolic solution as the remedy most likely to initiate restoration of the joint to health and usefulness. Of this description was the case of a man æt. 34 in St. Mary's Hospital last January. Shortly before Christmas, 1900, he had slipped and sprained his right knee, and he was admitted on January 11 with the joint greatly swollen and suffering much pain. The effusion showing no tendency to diminish, the joint was aspirated on the 15th, and fluid was withdrawn with the physical peculiarities already named. Notwithstanding this the pain continued and the swelling began immediately to return. The man himself was, moreover, in a very restless, irritable state, so that others in the ward said that he must be "off his head." His temperature was but slightly raised, reaching 100° F. on only two occasions. Accordingly, on January 18, the fluid was again removed from the knee, and the joint was then washed out with solution of 1 in 100 carbolic until the fluid returned perfectly unclouded and clean. From this time onward he began to improve both locally and constitutionally, and after

a course of massage and movement he was discharged on February 22 almost fit for work and with recovery in sight.

I mention this case in briefest terms alone, because I regard it as of the same class as that which is now to follow, and which afforded Dr. Poynton and Dr. Paine the opportunity for important investigations, which make it worthy of publication.

George H., æt. 14, admitted to St. Mary's Hospital on March 22, 1901, gave a history of having hurt his right knee shortly before Christmas, 1900. The knee had been knocked and immediately began to swell, but after a few days' rest the swelling had subsided, and he resumed work. Again, on March 15, he knocked his knee against the wall as he lay in bed, and this blow was immediately followed by swelling and considerable pain. Such was his own account, but a story in all probability more trustworthy was that of his grandmother, who said that he had been brought home on that day and put to bed because of shivering, sickness, and pains in the head. The knee, at any rate, was the most noticeable feature of his illness, and fomentations and rest being without influence, he was sent by his doctor to the hospital. The knee was then tightly distended with synovial effusion, and he was suffering much pain. Furthermore, he was obviously very ill. His temperature varied from 100° to 101° F.; he was restless and irritable, talkative, and inclined to wander, and with difficulty induced to take any food. The effusion showed no sign of diminution, and accordingly, on March 26, the fluid was removed by aspiration, it being our intention to wash out the joint with carbolic in case the effusion should return or the nature of it should suggest the desirability of this procedure. The fluid did display the naked-eye appearance above described; diplococci, some of them arranged in chains, were found in it, and the effusion did return. Except slight temporary improvement on May 27 and 28, the boy was no better: rather was he worse; his temperature was higher, and his mental and general condition showed no change for the better. On April 2, therefore, the knee was again aspirated and thoroughly washed out with carbolic lotion 1 to 100. Thereafter he began to mend in all respects, save that there was a slight return of fluid, and there was no setback from steady and continuous return to convalescence until he left the hospital on May 9. By Dr. Poynton's wish he had been treated with salicylates from April 13 onwards,

and there can be little doubt that this helped to promote recovery.

I now leave it to Dr. Poynton and Dr. Paine to deal with and record the results of their investigation of this interesting case. Apart altogether from what they will say, it is clear, as I have indeed always considered in dealing with them, that in these cases of arthritis we have to do with a state of things determined by the invasion of some infective micro-organism, and that the most rational and effective mode of treatment must be that by washing out the joint and the removal of all the deleterious products to which the organisms may give rise.

Bacteriological and Experimental Investigations by Dr. Alexander Paine and Dr. F. J. Poynton.

Upon March 26 the fluid obtained from the knee-joint was added to bouillon and incubated at blood-heat for twenty-four hours.

The fluid itself was slightly opalescent, and a film showed fibrin and a considerable number of polynuclear leucocytes. In the film there were numerous very minute diplococci in chains.

On March 30, 20 minims of the bouillon were injected intravenously into a rabbit. Upon the third day there was arthritis of the tarsal joint upon the left side. Later arthritis followed in other joints. The heart was evidently affected, a systolic murmur developed at the apex, and action became very excited.

The arthritis gradually but slowly commenced to subside, though when the animal was killed upon May 31st there was a good deal of thickening around the affected joints.

The necropsy showed much enlargement of the heart and dilatation of the left ventricle, with mitral valvulitis. The liver was engorged, the spleen small, the kidneys pale, and there was excess of clear fluid in the peritoneal cavity. There was no suppuration.

The exudation in the joints resembled in appearance unboiled white of egg, and much of the swelling of the joints was due to chronic inflammatory changes in the periarticular tissues. There were no bony changes, and there was no erosion of the cartilages.

Upon April 2 the patient's joint was aspirated a second time, and cultures made in bouillon and in a medium of milk bouillon and lactic acid.

Films of the joint fluid showed leucocytes, fibrin, and diplococci growing in chains. The cultures showed a pure growth in each tube.

Intra-venous injection of 1 c.c. of the bouillon culture into a rabbit produced again a polyarthritis. This time the rabbit was given salicylate of soda in two-grain doses by the mouth through a soft catheter.

The effusion completely disappeared from both knee-joints, but the carpal joints, which were affected later, did not subside. In addition to arthritis a doughy swelling about the size of a filbert developed upon the outer side of the right fore-limb. This swelling contained a clear sterile fluid. The necropsy showed no visceral suppurations or valvulitis.

Upon April 7 a subcutaneous inoculation into another rabbit was made from a culture in ascitic bouillon. A week afterwards a systolic murmur was detected, which lasted for three days and then disappeared. The animal completely recovered.

Upon April 24 an intra-venous inoculation of 2 c.c. of the ascitic bouillon culture was made into a fourth rabbit. Polyarthritis developed in the first week and subsided, the animal making a complete recovery.

This investigation demonstrates that the exciting cause of the arthritis in the second of the two cases described by Mr. Page was a diplococcus, and that this diplococcus resembled in its morphological and cultural characteristics the diplococcus of rheumatic fever, rather than the *Streptococcus pyogenes*. It is interesting that Mr. Page from the clinical aspect of the case, and we from the bacteriological, came quite independently to the conclusion that the arthritis was unusual in its type.

There are a considerable number of cases of arthritis which are somewhat vaguely termed "septic," in which it is natural to suspect a microbic infection, which will present some peculiar features.

At one end of the series there is a virulent suppurative arthritis, the result of infection with the *Streptococcus pyogenes*. At the other there is simple rheumatic synovitis, the result of infection with the diplococcus of rheumatic fever.

Such a condition as the one that is recorded in this paper lies somewhere between these two extremes. In the clinical

history it resembles in some respects the more septic forms of arthritis. The experimental and bacteriological results bring it closer to the rheumatic arthritis. The scientific importance of such cases as these turns upon the light they may throw upon the condition known as rheumatoid arthritis. The practical importance lies in the fact which Mr. Page has dwelt upon, that they are amenable to a form of surgical treatment which is not severe, and which we believe in this particular instance was assisted by the administration of full doses of salicylate of soda.

Another feature of interest is the great number of the diplococci that were present in the inflammatory exudation. We have found both from investigations on rheumatic fever in man and in animals that the acute and clear effusions of that disease are as a rule sterile, although the diplococci may be present in the synovial membrane beneath the endothelial lining. In this case the organisms were present in the exudation in numbers, and we should presume that the endothelial layer of the synovial membrane was considerably damaged.

Since this paper was received by the Society we have strengthened the view that such cases as those recorded to-night may throw light upon certain forms of rheumatoid arthritis. We have demonstrated in the exudation and synovial membrane of a knee-joint, showing all the changes of rheumatoid arthritis, a diplococcus which was isolated, and produced in ten weeks an osteo-arthritis of the right knee-joint of a rabbit. The investigation was laid before the Pathological Society of London in January. We show on this occasion this experimental osteo-arthritis, and for comparison a type of arthritis produced by the diplococcus of rheumatic fever. The effusion in each instance, it will be seen, is quite clear. We venture to lay especial stress on the clinical importance of these results, for we have here the two types of non-suppurative arthritis, namely, the osteo-arthritic, and synovial and periarticular, each produced experimentally from the respective conditions in man. Doubtless these two types merge into one another, but it can hardly fail that these results must in the future throw light upon rheumatoid arthritis. There is one point of clinical interest which concerns the occurrence of a monarthritis, a condition which some have found difficult to reconcile with an infective agent: a glance through the microscope at the exudation from Mr. Page's case (a monarthritis), and the evidence of this experimental osteo-arthritis—produced not by an injection into the

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DESCRIPTION OF PLATE IX.

To illustrate Mr. Page's two cases of Arthritis of the Knee.

A film of the exudation removed by Mr. Page from the knee-joint of Case 2.

There are many minute diplococci arranged in short chains and in masses lying in a fibrino-cellular network.

The especial features of interest are—

1. The great number of diplococci that were present in the exudation.
2. The grouping of diplococci in masses.
3. The minute size of the diplococci.

The diplococcus was morphologically and in culture indistinguishable from the diplococcus of rheumatic fever.

joint, but by an injection into the general circulation,—must, we believe, make us very cautious in separating at the bedside cases of monarthritis, however chronic, from the multiple joint infections.

It is unfortunate that at the time when we investigated Mr. Page's case we had not adopted the plan of preserving the joint exudations in sterilised pipettes, for the peculiar viscid exudation that we found in the joints of the rabbits is a point of much clinical interest. Triboulet has clearly demonstrated that such exudations in man may give rise to perfect fluctuation, yet no fluid be withdrawn on aspiration; and this particular type of viscid exudation may be an important indication of a commencing chronic arthritis.

Finally, the last and most difficult clinical problem is the respective parts that must be assigned to the infective agent and the tissue resistance in these various types of arthritis. The clinician will possibly give the second place to the micro-organism, the bacteriologist, perhaps, will do the reverse; but the occurrence in these rabbits of various types of arthritis produced by diplococci, morphologically and culturally indistinguishable, but isolated from various types of arthritis in man, should make us cautious in arriving at any hasty conclusion upon this point.

XVI.—*A case of Malignant Endocarditis giving Widal's Reaction.* By W. Hale White, M.D., and W. C. C. Pakes. *Read March* 14, 1902.

Jane M., æt. 17, was admitted into Guy's Hospital under Dr. Hale White on June 6, 1901. She had suffered for some time from anæmia; she had never had rheumatic fever, chorea, or scarlet fever. She was comparatively well till June 1, when she felt "out of sorts," and she had gradually been getting more ill since then. On arrival at the hospital it was found that her temperature was 103°, pulse 120, and she was obviously ill. There was slight œdema of both legs; the cardiac impulse was one inch external to the nipple, and there was a murmur indicative of mitral regurgitation. It was thought that she had malignant endocarditis. She was so anæmic that the possibility of some primary form of anæmia was discussed, but the examination of the blood did not support this view, and the ophthalmoscope showed the fundus to be healthy. Typhoid fever was also thought of, especially as the spleen was enlarged, but there were no spots.

There was no alteration of symptoms until September 9. From admission till then she lay in bed, obviously very ill, with irregular pyrexia, the temperature usually ranging about 102° and 103°. Her pulse was rapid, she became more and more anæmic and gradually weaker. The murmur was unaltered, and it was soon clear that we had to deal with a case of malignant endocarditis.

Three different strains of antistreptococcic serum were tried, and the trial of each was at least a week, but none had any effect on the course of the disease; therefore the use of the serum was discontinued.

On September 9 she became hemiplegic on the left side of the body; from this date she rapidly became worse, and she died on September 13.

The post-mortem, made on September 14, showed malignant endocarditis of the mitral valve, with softening of the right central ganglia of the brain. The spleen was soft and large. The rest of the body was healthy.

We do not record this case on account of the symptoms,

for it was a straightforward example of malignant endocarditis terminating in a usual way; nor on account of the failure of antistreptococcic serum to control the disease, although, as will be mentioned directly, streptococci were easily found in the blood. It will be noticed that antistreptococcic serum from three different sources was tried, for it is possible that clinically sometimes one strain may be effective when another is not, but none were of benefit; and this accords with our previous experience that very little reliance can be placed on antistreptococcic serum in this disease. But the case is recorded on account of the bacteriological findings, and because of their great clinical interest.

The following is the account of the bacteriological examination undertaken in connection with this case:

On June 10 a specimen of the blood taken from the ear was examined for agglutination. It was tested with the strain of the *B. typhi abdominalis* in the Guy's Hospital laboratory known as "A," but failed to show agglutination, even in a strength of 1 in 2.

On June 21 specimens of blood from the ear were examined, first for agglutination and secondly for streptococci. Agglutination was produced with strain "A" when the serum was in a strength of 1 in 2 and 1 in 20, but was not produced in a strength of 1 in 200.

One specimen of blood, containing about ½ c.c., was collected in a pipette, and about 1 c.c. of sterile broth was added to it, the whole being incubated at 37° C. for twenty-four hours. At the end of this time microscopic examination showed that there was a luxuriant growth of streptococci. Subcultures on various media showed that this organism conformed to the description of the *Streptococcus longus*, and that no other organism was present.

On June 29 a catheter specimen of the urine was obtained under strictly aseptic precautions, and examinations and cultures were made. The microscopic examination revealed the presence of two organisms, one a motile bacillus and the other a streptococcus. Agar plates were made directly from the urine, and the two organisms isolated. The streptococcus upon subculture proved to give identical reactions with that isolated from the blood on June 21. The bacillus belonged to the paracolon group, clotting milk in three days. Every attempt to isolate the *B. typhi abdominalis* failed, and we feel

sure that there were only the two above-mentioned organisms present.

On July 7 further samples of blood were obtained from the ear, and were treated exactly as were those collected upon June 21. In the $\frac{1}{2}$ c.c. of blood no streptococci could be found.

The agglutination test was tried with five strains of the *B. typhi abdominalis*, named respectively "A," "3," "4," "5," and "Firth." The result of this examination is tabulated hereunder:

Strain of bacillus.	Serum, 5 per cent. (1 in 20).	Serum, 0·5 per cent. (1 in 200).
A	+	$\frac{\pm}{2}$
3	+	0
3	+	0
4	+	0
5	+	0
Firth	0	0

(The sign + means that there was complete agglutination within half an hour; $\frac{\pm}{2}$ means that there was a partial reaction within half an hour, *i. e.* many individual bacilli were free in the fluid, although there were many clumps of motionless bacilli; 0 means that there was no effect upon the bacilli.)

It will therefore be seen that in a strength of 1 in 20 four out of five different strains of the *B. typhi abdominalis* were agglutinated by the serum of this patient.

On September 13 the patient died, and the post-mortem examination was made on the 14th.

Examinations were made of the blood from the right and left ventricles and of the spleen.

From the blood collected from the ventricles the *Streptococcus longus* was obtained in pure culture.

From the spleen the same streptococcus was isolated, and the *B. coli communis*. All attempts to isolate the *B. typhi abdominalis*, however, failed.

Thus the only evidence that the case was one of enteric fever was that the patient's serum reacted with four different strains of *B. typhi abdominalis* during one period of the disease.

The fact that on the first occasion the serum gave no reaction, and that on a subsequent occasion a reaction was obtained, suggested that the patient might have acquired

typhoid fever subsequent to, and in addition to her malignant endocarditis; but it will be noticed that examination of the urine during life, and of the blood of the spleen after death, negatived this view, nor were any spots ever seen, nor were there any indications of healed typhoid ulcers in the intestine. It is well known that patients may have typhoid fever without intestinal ulceration and without spots, but in the face of the negative examination of both blood from the spleen and urine there is, we consider, no doubt whatever that this patient had not had typhoid fever.

Thus we have an undoubted case of a patient suffering solely from malignant endocarditis of streptococcal origin, and yet, during the progress of the disease, she developed in her blood-serum certain substances which had the power of—in a strength of 1 in 20—agglutinating the *B. typhi abdominalis*. This is of considerable importance, not only in considering in any case the diagnosis between malignant endocarditis and typhoid fever, but also because one of us has examined many cases of ulcerative endocarditis and, like many authors who have published their results, has hitherto failed to obtain a Widal reaction in this disease.

Before leaving the subject we would point out that the presence of the Widal reaction in this case cannot be due to the fact that the patient had had typhoid fever at some time previous to her coming to Guy's Hospital, for she did not give the reaction when first admitted.

XVII.—*A case of Acute Paralytic Dilatation of the Stomach.* By A. E. Sears. *Read March* 14, 1902.

THE following is an account of an illness which occurred in the early part of last year, and which confined me to bed for a period extending over two months.

The course of the illness may be divided into three periods:

1. The onset and period of intermittent fever with fæcal accumulation, constipation, and loss of flesh.
2. The period of phlebitis of the left leg and iliac region with continued loss of flesh.
3. The acute dilatation of the stomach and subsequent recovery.

Family and personal history present no points of interest, but it may be mentioned that I have never at any time of my life suffered from dyspepsia or constipation of a chronic character.

The onset of the illness occurred insidiously during the last two or three weeks in January; a feeling of malaise and occasional shivering fits in the afternoon, with some profuse perspiration at night, made me take to my bed on February 4, when my temperature was found to be 102·4°.

My two partners, Dr. Walter Kidd and Dr. Mackern, on the following day made a very thorough examination, but beyond the rise of temperature nothing abnormal was found. The chest was free from adventitious sounds, there was a complete absence of any abdominal tenderness or distension, the spleen was not enlarged, nor were there any typhoid spots. The urine was high-coloured, and contained a small deposit of urates.

From February 4 until the 17th of that month the temperature was of an intermittent character, rising from normal in the morning to 102°—103° during the afternoon.

The condition during these two weeks was one of comparative comfort from the early morning until midday, after which time pyrexial pains in the limbs and head commenced, and a feeling of chilness followed, with occasional delirium, which continued until five in the evening or later, when the

temperature seemed to have reached its maximum and profuse perspiration accompanied the decline of the fever. The motions were very constipated, and there was evidently an accumulation of fæcal matter in the lower bowel, though this could not be definitely felt through the abdominal wall.

A variety of purgatives were administered, and the resulting actions varied considerably in quantity and in character; at one time a copious, dark-coloured, and offensive motion would be passed, as if it had lain in the bowel for some considerable period, while at other times only a very small constipated and light-coloured motion would result.

The diet at this time consisted almost entirely of milk and beef tea.

Although there was progressive loss of flesh and strength, yet no other signs or symptoms were noticed until the second stage of the illness occurred, viz. the phlebitis, the first indication of this being a pain in the left groin which occurred when at stool. Pain and extreme tenderness to touch became rapidly worse, and spread gradually down the thigh to the foot and also up into the left iliac region.

At this time, although the superficial veins were acutely tender, and could be distinctly seen and traced down the leg, yet there was hardly any swelling, and certainly no œdema of the parts, and it may be of interest here to state that œdema of the leg did not occur until convalescence had commenced. The painful period of the phlebitis lasted about another two weeks, during which time the temperature became somewhat irregular, but commenced to descend at the end of ten days, and fell to below normal on March 1.

The condition of the bowels had improved, although occasional large and offensive motions were passed.

The diet now consisted of milk, eggs and milk, Benger's food, and beef tea, with small doses of brandy, but in spite of the increased amount of food loss of flesh and strength continued, and especially so toward the latter part of these two weeks.

As no discomfort was experienced after food the diet was further increased by the addition of a lightly poached egg.

Having now arrived at the third stage of the illness, viz. the acute dilatation of the stomach, I will endeavour to give the history of its onset and course as fully as possible.

It has already been mentioned that no discomfort was experienced after the increased diet on March 3 and 4, and on the following day two poached eggs were allowed, but

after this meal digestion did not seem to take place so easily as on the previous occasions.

The feeling of nausea and discomfort soon gave place to a dull and continuous pain in the epigastrium, the abdomen became distended, and as no relief was experienced by way of the mouth or rectum the diet was reduced to small quantities of milk and beef-tea.

On the following day the symptoms increased in severity, and on palpation of the abdomen there was some splashing in the region of the umbilicus, which was then considered to be due to fluid in the bowel. The note on percussion over the abdomen varied, but in no position except in the epigastric region was it of a tympanitic quality.

The general and local conditions became worse, and with an increase of pain came an increase of distension, which now seemed to have invaded the whole of the abdomen, and though several enemas of soap and water were administered the distension progressed.

To these symptoms were added a feeling of intense nausea with distress, and later thirst, to relieve which a small quantity of cold water was taken, but this seemed to aggravate all the troubles and to cause, in addition, acute stabbing pains in the left hypochondriac and lumbar regions.

From this time until the evening the symptoms increased in severity, when a condition of collapse occurred, the temperature falling to 97°, and the pulse becoming very soft and running in character.

The abdomen by this time was greatly distended, especially in the epigastric region, and a splashing sound could be elicited from any part between the ensiform cartilage and pubes, and on percussion a dull note was obtained below the level of the umbilicus, above which it was tympanitic. The whole surface of the abdomen was uniformly distended, and no peristaltic waves were observed.

During the day and, in fact, for forty-eight hours, very little nourishment had been taken, certainly the quantity did not exceed 15 oz. in all.

Several unsuccessful attempts at vomiting had been made at different times, but the stomach seemed quite unable to relieve itself by this route, and after a final attempt had been made in the knee and elbow position, it was decided to withdraw the contents by the stomach-tube.

A Jaques' No. 18 œsophageal tube was accordingly passed, and three quarts of fluid were syphoned away.

Immediate relief was experienced; the intense pain in the small of the back, and the dull, heavy, dragging pain in the abdomen vanished with the removal of the fluid.

The contents of the stomach, which, as has already been mentioned, measured three quarts, consisted of a dark green sour-smelling fluid, with a large quantity of mucus and a small sediment of much the same character as that found in beef tea.

On examination of the abdomen soon after, the stomach by percussion was found to have receded to a point about three fingers' breadth below the level of the umbilicus, and the succussion splash could not be elicited.

The treatment adopted consisted of 4-oz. nutrient enemas with brandy alternating with 5 oz. warm saline solution and a hypodermic injection of gr. $\frac{1}{60}$ of strychnine every four hours. All feeding by the mouth was discontinued for twenty-four hours, but after that time a teaspoonful of equal parts of beef-juice and water were given every half-hour.

The amount of urine passed diminished considerably during the period of dilatation, but there was never a complete suppression.

There was no action of the bowels for five days, the copious water enemas which were given for this purpose being often entirely retained.

With the exception of a very intense thirst, the comfortable condition continued for three days, when the early symptoms of dilatation again occurred, indicated by a feeling of weight in the epigastrium, and an increase of dulness on percussion with succussion splash, and the stomach was again emptied and washed out with a solution of hyposulphite of soda. At the end of another four days this process was again repeated, and the general condition had so improved by this time that it was decided to pass the œsophageal tube every day. The daily emptying and lavage were attended with most beneficial results, for the primary withdrawal of fluid, which consisted entirely of stomach contents, decreased rapidly, and the colour, odour, and amount of *débris* diminished in proportion. The diet by now consisted almost entirely of pulped beefsteak, cream, and small quantities of brandy and water, and at the end of another week it was deemed only necessary to wash the stomach out on alternate days.

A temporary disturbance later was caused by some seakale which had been taken with the pulped meat, but this

was at once relieved by washing out immediately. The only return of the acute dilatation followed the trial of some unpulped meat at the end of the third week in March, some two and a half weeks from the commencement of this stage of the illness, and in this case the stomach was not washed out until sixteen hours after the meal had been swallowed, as the symptoms were not very distressing.

The result of this lavage showed that the meat had been very little altered during its stay in the stomach, but it was evident that it had caused some irritation, as discomfort and a degree of dilatation followed the ingestion of any food, and it was found desirable to discontinue all nourishment and fluid by the mouth for thirty-six hours, during which time thirst became almost intolerable.

At the conclusion of this fast the contents withdrawn from the stomach measured over two and a half pints of a dark green coloured fluid presenting much the same characters as before.

For a week or more the tube was passed daily, but at the end of that time the general and local conditions improved so rapidly that before long its use was entirely discarded, and a change to the sea-side completed the cure.

Remarks.—Up to the present time fifty-two cases of this disease have been collected. In 1898 Box and Wallace reported a series of twenty-three cases to this Society, which are incorporated in vol. xxxi of the *Transactions*. Eighteen of these ended fatally, and out of the remaining five which recovered three do not appear to have been of a very serious character.

Quite lately Campbell Thomson, in a paper read before the Royal Medical and Chirurgical Society, added to the collection just mentioned another twenty-nine cases, making in all fifty-two.

Of these forty-four were fatal; of the remaining eight which recovered mention has been made of five. In the additional three acute dilatation occurred in two after operation, particulars of which are to be found in Mayo Robson and Moynihan's work on *Diseases of the Stomach and their Surgical Treatment*, and the other case was reported in the *Philadelphia Medical Journal* of March, 1901; in this case acute dilatation followed an indiscretion of diet in a man æt. 41. In this instance the abdomen was opened and the stomach punctured and drained.

A degree of interest is attached to the case which I

have just read by the absence of vomiting, which in all previously reported cases, whether ending fatally or otherwise, was a constant symptom, and in the majority a very distressing and important one. In the account of the fifty-two cases vomiting, generally profuse and persistent, was certainly present in thirty-four, in one other case it was described as slight, and in the remaining seventeen there is no mention of this symptom, but it most probably was present. In my case vomiting was never present, nor was it ever possible to relieve the stomach in this way; it should be remembered that the condition of the body then was one of extreme emaciation, and the abdominal, like the other muscles, were very wasted.

The nature and cause of the period of intermittent fever in the first stage of the illness has not yet been satisfactorily explained. The diagnosis at the time rested between influenza, which was then very prevalent in the neighbourhood, and a septic absorption from the fæcal accumulation.

The possibility of typhoid fever cannot be absolutely excluded, but, with the exception of the temperature, it was considered that there were not sufficient signs or symptoms to warrant the diagnosis, but I much regret that I did not submit the blood to Widal's test. The occurrence of phlebitis may seem to support the diagnosis of typhoid, but it has also been suggested that the inflammation of the veins was due to the accumulation of fæcal matter in the bowel.

A case recorded by Schult, and which ended fatally, bears many points of resemblance to mine. It is reported as follows:

The patient was a young woman who, having been previously strong and healthy, was taken with a vague fever in August, which resisted treatment and reduced her to a condition of extreme emaciation. The temperature became normal in November, but the marasmus was still extreme. With careful feeding and nursing she improved steadily. After two weeks she took food with relish, and had no pain or nausea.

In December, some three weeks later, she complained of an intense abdominal pain, and commenced to vomit, and the vomiting persisted until death, which took place three days later.

The post-mortem examination showed an enormously distended stomach.

It may be remarked that in this case visible peristalsis

of the stomach was noted, a condition, I believe, not before or since observed in cases of acute dilatation.

Several causes have been assigned by various writers to account for the dilatation. By some the excessive secretion has been thought to be the primary factor, and this view was probably held by Fagge, who reported the first four cases in the *Guy's Hospital Reports* for 1873.

A spasmodic contraction of the pylorus has been suggested, but has not found much favour.

Another theory is that of a mechanical obstruction, caused by constriction of the intestine by the superior mesenteric artery.

Again, others have considered that the most probable explanation is a primary paralytic condition of the stomach walls, and later, that the distended organ cannot discharge its contents into the intestines on account of the actual pressure which it exerts on a particular portion of the duodenum.

These theories have been fully discussed in the paper by Campbell Thomson which has been previously mentioned, and though I naturally do not feel competent to express an opinion of any worth on them, yet I would suggest that in my case it appears probable that some septic condition of the bowel may have produced toxins which, acting generally on the nervous system, and more particularly on the nerves of the stomach, caused a condition of gastric atony, and that it only needed the introduction of some indigestible article of food to start the dilating process.

And I would further suggest that the secretion occurred with the dilatation, and later the excessive amount of the secretion favoured and advanced the dilatation.

May I finally add a few words on a portion of the subject upon which I can speak with more knowledge and confidence, viz. the treatment?

This resolves itself mainly into (1) removal of the excessive secretion, and (2) introduction of a suitable form of nourishment.

As regards the latter, rectal feeding may be necessary for a short period at first, though there is the danger that the bowel will not tolerate, even if it absorbs this form of nourishment. Pulped meat, *i. e.* meat thoroughly minced and passed through a wire sieve, agreed most perfectly in my case; large quantities of this could be taken with ease and comfort when the stomach was unable to digest anything

else, and it seemed to be almost completely absorbed from the first, judging from the stomach washings and also by the progress which was made when the diet consisted solely of this.

Certainly liquids should be reduced to the smallest possible quantity, and the thirst (which was a very distressing symptom in my case) relieved by warm saline rectal injections and small drinks of very hot water. The water should be as hot as can be swallowed, it then having the double advantage of being more satisfying and not so quickly consumed. Beef juices and meat extracts increased the thirst, and did not seem to be well tolerated.

Subcutaneous injections of strychnine are very useful, and this form of medication seems to be the only available one.

XVIII.—*Further Report upon a Case of Lymphadenoma with Growths in the Skin.* By Lee Dickinson, M.D., *and* W. J. Fenton, M.B. *Read March* 14, 1902.

At the meeting of this Society on April 26, 1901, a boy æt. 16 was shown as a case of general lymphadenoma with secondary growths in the skin, a short account of whom will be found in vol. xxxiv *Clinical Transactions*, p. 246. Some difference of opinion as to the true nature of the case was expressed, and a further report as to its progress was asked for. Unfortunately there is not much to add to the clinical account previously given, and the report now furnished is mainly concerned with the post-mortem appearances. The suggestion of antisyphilitic treatment was adopted, in the form of iodide of potassium, without, however, any beneficial effect.

The boy remained fairly active for a time, but several weeks before his death he became bedridden owing to progressive anæmia, emaciation, and abdominal enlargement. A blood-count at the middle of June showed 1,200,000 red and 33,200 white cells per cm.; no relative increase of lymphocytes and no myelocytes.

No great change occurred in the lymphatic glands in the neck, axillæ, or groins, and the only change in the skin growths was a sluggish further involution. During June and July the liver and spleen enlarged rapidly; ascites came on, together with œdema of the scrotum and lower extremities, and on several occasions the abdomen had to be tapped. The temperature became increasingly irregular, generally showing a difference of two degrees between morning and evening, and occasionally reaching 102° in the evening.

Cerebral symptoms were entirely absent, and death resulted from exhaustion on August 10.

At the post-mortem examination the most important morbid appearances were as follows:-

Body extremely emaciated.

In the skin of right pectoral region were four yellowish tuberculated areas varying in size from that of a sixpence to that of a florin.

Moderate œdema of lower extremities; abdomen distended with clear ascitic fluid.

Lymphatic glands.—In addition to the enlargement of the cervical, occipital, axillary, and inguinal groups which had been obvious during life, the pelvic and lumbar glands were found to be greatly affected. They varied in size from a marble to a pigeon's egg, and were embedded in well-formed fibrous tissue, by which the great vessels were surrounded if not compressed. On section these glands were firm, pale, and homogeneous, and neither here nor elsewhere was there the slightest sign of suppuration or caseation. The mesenteric glands were distinctly enlarged, but somewhat softer and more vascular than the lumbar; those in the portal fissure were not enlarged. In the anterior mediastinum, at the point of reflection of the pericardium from the base of the heart, the glands formed a firm mass of growth, but the site of the thymus body was normal.

The spleen was five or six times the natural size, and weighed 1 lb. 9 oz. It contained numerous masses of growth scattered throughout, of rather lighter colour than the splenic pulp, but for the most part reddish rather than yellow or white. The typical " hardbake " appearance was absent.

Liver large, weighing 4 lbs. 5 oz., firm and pale, but not otherwise abnormal to the naked eye. The other abdominal organs call for no remark.

In the thorax was a mass of firm, whitish growth, as large as a walnut, springing from the periosteum of the left fourth rib, close to the spine. The lungs were free from any sign of tubercle or morbid deposit, and the heart was healthy except for slight dilatation of its cavities.

In the cranial cavity, attached to the inner surface of the dura mater, were three small growths of similar character to that on the rib, one in the right middle fossa, one at the vertex near the longitudinal sinus, and one at the junction of the tentorium and falx cerebri.

The bones in general were not examined, but as the heads of the tibiæ were slightly expanded, and had been tender during life, a vertical section was made of the left tibia. This disclosed three circumscribed, yellowish, slightly softened areas in the cancellous tissue—two in the head, just above and just below the epiphysial line respectively, and one in the shaft. The latter was at the spot where a slight prominence on the anterior surface of the bone had been felt during life, and had given rise to the suggestion of a syphilitic node.

Bacteriological cultures from the fresh spleen and from some of the softer lymphatic glands were taken by Dr.

Charles Slater, and resulted in the growth of streptococci, which, however, is nothing new.

All the diseased parts were examined microscopically, and with the exception of the liver they all show the same kind of change, namely, a highly cellular lymphoid growth with a fibrous reticulum of varying density, most dense in the glands and periosteal growth, and least dense in the bone. In the spleen the coarser trabeculæ enclose areas of small round-cells with but an obscure reticulum, but without giant-cells or any sign of degeneration.

Many lymphatic glands and the spleen were carefully stained for tubercle bacilli, with a negative result.

When the liver came to microscopical examination it unexpectedly proved to be decidedly lardaceous, both in anatomical structure and in its coloration with methyl-aniline violet. But the important point about the liver is that one of the sections disclosed a small tuberculous focus, caseous in the centre, with well-formed giant-cells at the edge, and containing a few indisputable tubercle bacilli. This little focus is evident to the naked eye upon the slide, but in the post-mortem room it was probably just upon the limit of naked-eye vision; at any rate it escaped detection, and there may have been others in the liver, though certainly there were no others in the many small portions preserved for the microscope.

The lardaceous condition of the liver does not seem to have any essential bearing upon the case, but is worth remarking, because at one time there existed some idea of a relationship between lardaceous disease and lymphadenoma, and when Sir Samuel Wilks opened the discussion on lymph-adenoma at the Pathological Society in 1878 he suggested the question of that relationship as one of the points for discussion. However, although lardaceous disease was much commoner then than now, none of the many contributors to the debate even touched upon this point, which is surely a sufficient proof that there is no real connection between lardaceous disease and lymphadenoma.

The two great causes of lardaceous disease are, of course, suppuration and syphilis, but a third occasional cause seems to be almost admitted, namely, profound cachexia of different origin,—from malaria, for example. This patient escaped both suppuration and syphilis, but for some months before death he had been in a state of progressive enfeeblement and anæmia, ending in as profound a cachexia as ever results from suppuration, syphilis, or malaria.

It is to be regretted that the final test of tuberculosis, inoculation experiments upon guinea-pigs, was not employed, but looking at the whole case—its clinical course, especially the fluctuations in the size of the glands and the remarkable involution of the cutaneous tumours, the post-mortem appearances, and the failure of the microscope to find tubercle or tubercle bacilli anywhere except in the liver,—we conclude that the essential disease was lymphadenoma, and that the limited tuberculosis of the liver was merely a secondary infection.

Of syphilis there was no evidence whatever.

P.S.—Since the above report was written, Dr. F. W. Andrewes has most kindly examined our microscopical sections, and although, in his own words, he gives only "a tentative opinion on a difficult case," we feel that his opinion is too valuable not to be quoted.

Upon the whole Dr. Andrewes confirms our view that "the case is one of lymphadenoma upon which tubercle has supervened." The gland from which we submitted sections is apparently "affected by the hard form of lymphadenoma, with much diffuse fibrosis and endothelial hyperplasia, but without the characteristic large cells, which makes the diagnosis a little uncertain." He points out that the masses in the dura mater and skin reproduce the characters of the gland; and that in the spleen, although the fibrosis reaches an unusual degree, here also the condition is probably the hard form of lymphadenoma. The liver is, of course, tubercular, but elsewhere Dr. Andrewes found nothing resembling tubercle except some rather suspicious areas of early caseation and obscure giant-cells in the growth from the rib.

XIX.—*Cases illustrating the Mode of Origin of certain Cysts of the Neck.* By T. CARWARDINE, M.S. *Read April* 11, 1902.

I WISH to bring to your notice certain cysts of the neck, and to indicate their bearings on classification.

The significance of the thyro-glossal duct of His in its relation to cysts is now well known, and we may take this as a standpoint. Cysts and fistulæ arising from this duct usually have a pedicle which passes through the thyro-hyoid membrane in the middle line just below the hyoid bone, between the body of the hyoid and the thyroid cartilage. They appear to be connected with the second visceral cleft, immediately behind the second or hyoid arch, and may be termed *thyro-hyoid cysts.*

In December, 1899, there came under my care a man æt. 31, who was deaf and dumb and one of twins, with a fluctuating superficial median tumour of the neck overlapping the episternal notch and manubrium sterni, and a hard cord could be traced upwards from it in front of the cricoid cartilage. I thought this would turn out to be a terminal cyst of the thyro-glossal duct. But on careful dissection at the operation the pedicle was found to pass through an interval in the cervical fascia into the middle of the crico-thyroid membrane. It was clearly a cyst arising from a duct-like pouch through that membrane, and I should propose to call it a *crico-thyroid cyst* (Fig. 3).

It may be mentioned that there is normally a small foramen in this position in the fœtus, and a small congenital intra-laryngeal cyst has been recorded at this site, but on the deep aspect of the crico-thyroid membrane.

Seeing, therefore, that both thyro-hyoid and crico-thyroid cysts were now established, it occurred to me that there should be a similar and homologous cyst in the space between the lower jaw and the hyoid bone, and after waiting some two years I happened to meet with the case in question.

The patient was a Jewish lad, æt. 12, who had noticed a lump in his throat for six months, in the middle line beneath

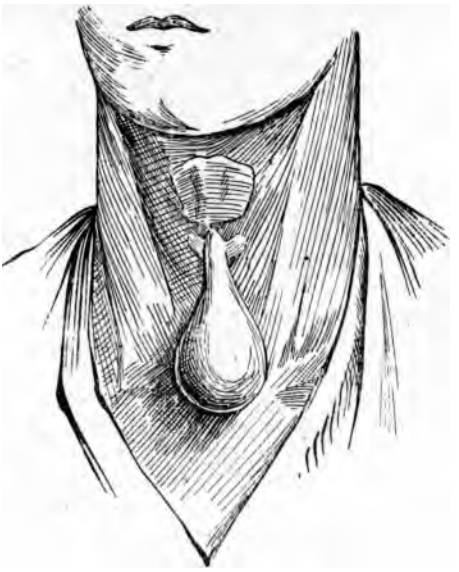


FIG. 3.—Crico-thyroid cyst.

FIG. 4.—Hyo-mandibular cyst (hyo-glossal).

the chin. On careful palpation a pedicle could be felt running deeply above the hyoid bone into the hyo-mandibular space. The diagnosis of a *hyo-mandibular cyst* proved to be correct at the operation. The cyst contained colloid material, had a soft, pulpy lining, and a hollow pedicle passed through an interval between the mylo-hyoid muscles and admitted a probe for about half an inch into the base of the tongue (Fig. 4).

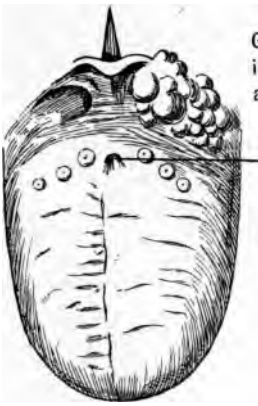
We thus have three median cysts: hyo-mandibular, associated with the first cleft; thyro-hyoid, associated with the second cleft; and crico-thyroid, in the succeeding space, which I shall refer to as the representative of a third cleft.

We now have to deal with the lateral aspects of the clefts and structures arising from them. The first case bearing upon these aspects is one of cystic hygroma.

The patient was a baby a few weeks old, emaciated, and having a large translucent *cystic hygroma* on the left side of the neck, hanging like a loose bag over the upper chest. A cyst like a ranula projected from under the tongue. The child soon died, and I was able to make a careful dissection. The hygroma occupied all the available space between the lower jaw and the heart on the left side and beneath the left clavicle. It was much convoluted, and contained a straw-coloured fluid. The sterno-mastoid and sterno-hyoid muscles were stretched over it in front, and everything behind was normal, including the larynx, trachea, and the thymus and thyroid glands. There was a small foramen in the crico-thyroid membrane, and the sacculus laryngis was perfectly normal. The cyst was adherent to the pericardium below, and passed into the substance of the left mylo-hyoid muscle above (Fig. 6). The item of the chief clinical interest, however, is the fact that the upper part was distinctly continuous with multilocular cysts at the left base of the tongue, embedded in whitish scar-like tissue. The foramen cæcum was independent and about $\frac{3}{16}$ of an inch deep, and the raphe was to the right of the middle line (Fig. 5). There could be no doubt that the hygroma and the cysts of the tongue were definitely related. The original sketch seems to indicate that the upper end of the hygroma was in front of the stylo-hyoid ligament, but I am unable to verify this, having no note of the fact, and owing to the specimen having unfortunately been mislaid. The hygroma seems to have originated in a lateral diverticulum of the first or hyo-mandibular cleft. In a similar case, recorded by Sir Frederick Treves some years ago, the origin was possibly from the second branchial cleft.

FIG. 5.

Sacculus laryngis, perfectly normal, without connection with the hygroma.

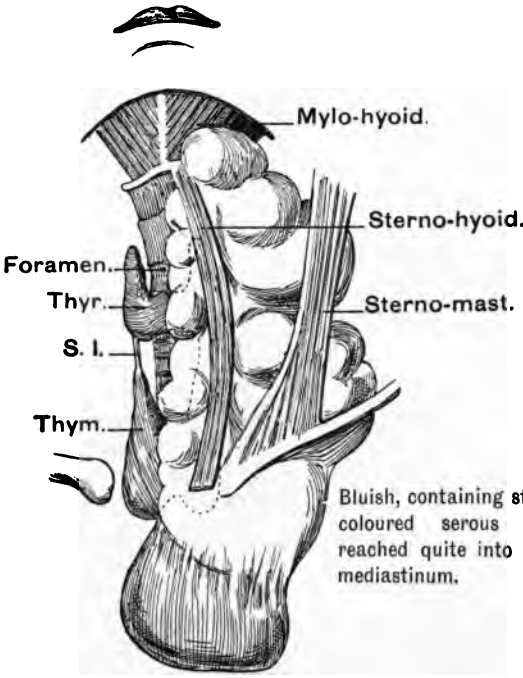


General cystic condition in a whitish scar-like area.

Foramen cæcum depressed, conical, $\frac{3}{16}$ inch deep, ending blindly.

Raphe to right of middle line.

FIG. 6.



FIGS. 5, 6.—Cystic hygroma connected with cysts at left base of tongue.

I have met with two other cysts of apparent origin from the first branchial cleft.

A young man was thought to have a soft caseous gland below the left angle of the lower jaw, from which cheesy material escaped at the time of operation. The tumour was said to have been growing for twelve months, and was about the size of half a hen's egg. Microscopically it proved to be a *chondro-adenoma;* the cartilage-cells were in a hyaline matrix, and the alveoli were lined in places by a single layer of columnar or cubical cells, and in the other parts the alveoli were packed with cells, but had the aspects of non-malignancy.

Another case was that of a *dermoid cyst* of the whole of the first branchial cleft, containing countless small soft spherical bodies like those in the hard roe of a fish; and at a second operation tubercle bacilli were found in addition. With this cyst there were associated what I may call a *hyo-mandibular tubercle* and a *supernumerary antitragus.* The original drawing is preserved, and is passed round, but, as the case was published at greater length some years ago, I must refer to the *Bristol Medico-Chirurgical Journal* for details.

The last case I will mention was that of a man æt. 33, whom I thought at first to have a pharyngeal diverticulum. A swelling had been noticed on the right side of the throat for two years, and it was said to have become gradually lower during that time (Fig. 7). It was doughy, the size of a pigeon's egg, and had deep connections above. Two years before I saw him he had been treated for what was said to have been hæmoptysis. He complained that about every three weeks or a month he felt a tickling sensation in the right side of his throat, followed about two hours afterwards by a fit of coughing by which he brought up phlegm and blood which smelt badly. When he coughed each night, as was usual, he brought up all his food. On laryngoscopic examination I was struck by a double condition of the sinus pyriformis, an appearance which I am informed is very rare (Fig. 8). I would therefore suggest that this association with the cyst of the neck was more than mere coincidence, and suggests an origin as a *diverticulum of the sinus pyriformis,* and thus from the third or fourth visceral cleft.

Now the foregoing cases seem to have an important bearing upon the classification of certain cysts of the neck, and also upon the true embryological positions of the thyroid and cricoid cartilages. A more detailed table is appended, but the following are the main features:

In the middle line of the neck we have certain cysts—hyo-mandibular, thyro-hyoid, and crico-thyroid,—alternating with the recognised visceral arches above, the mandibular and the hyoid; and below with what I would submit to be,

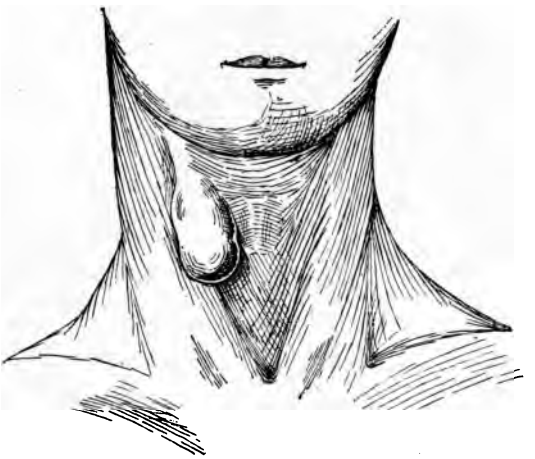


FIG. 7.—Lateral cervical cyst.

from clinical evidence, the representatives of successive arches, the thyroid and cricoid cartilages. Admitting these points as facts, we have an homologous series of *median* cysts, and we place the thyroid and cricoid cartilages in consistent

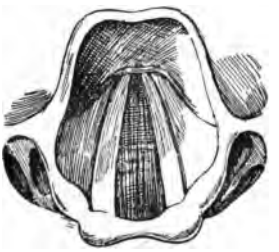


FIG. 8.—Double sinus pyriformis.

developmental positions. Moreover, above the first arch we have the stomodæum; and below the cricoid, the cartilages of the trachea, and a median pouch—that of the Emu,—figured in Mr. Bland-Sutton's book on tumours.

With these median diverticula there are associated *lateral* diverticula of the clefts, which give rise to various tumours and organs, some of which are indicated in the table.

From the first cleft we have, for instance, a cystic hygroma amongst other tumours of interest. From the second cleft it is possible that Treves's case, already mentioned, arose. Indeed, such an origin, amongst others, was suggested in the original description of the dissection. From the third cleft we have the lateral lobe of the thymus gland, and from the fourth cleft the lateral lobe of the thyroid gland. Still lower in the scale we have an essentially glandular organ, the lung.

Thus a rational classification would be into median and lateral, corresponding to the visceral clefts, and alternating with the visceral arches (*see* Table).

The general plan of construction in the neck—of a median diverticulum tending to reach the surface and paired lateral diverticula of glandular functions—appears to have its analogy elsewhere,—in the genital passage and ovaries, and in the allantoic vesicle and the kidneys. We might even speculate upon the relation of the umbilical vesicle to the liver and pancreas, but such imaginations as this would lead us into a new field of poetical physiology.

Table illustrating the Mode of Origin of Certain Cysts of the Neck.

NOTE.—Structures marked * are referred to in the text.

Arches and clefts.	Lateral.	Median.	Lateral.
1st Mandibular ARCH	Meckel's cartilage, etc.	Mandible	Meckel's cartilage, etc.
1st Hyo-mandibular CLEFT	*HYO-MANDIBULAR TUBERCLE with SUPERNUMERARY ANTITRAGUS Branchial epitheliomata and "epitheliomatous cysts"	*HYO-MANDIBULAR CYSTS *DERMOID OF BRANCHIAL CLEFT Median sublingual dermoids	*CYSTIC HYGROMA with basal lingual cysts. *CHONDRO-ADENOMA. Lateral sublingual dermoids.
2nd Hyoid ARCH	Palato-glossal arch, etc.	Body of hyoid bone.	Stylo-hyoid ligament, etc.
2nd Thyro-hyoid CLEFT	Branchial epitheliomata and "epitheliomatous cysts" ? Pharyngeal diverticula (Sutton)	*THYRO-HYOID CYSTS (arising in thyro-glossal duct) *Unpaired portion of thyroid gland	* ? Cystic hygroma (Treves's case) (Trans. Path. Soc. Lond., xxxii, 195). Godlee and Bucknall's case (a narrow pedicle passed through thyro-hyoid membrane [Brit. Med. Journ., 1901, i, 1204], here called a "pharyngeal pouch") (see below). ? Barker's case (Brit. Med. Journ., 1901, i, 1204).
3rd Thyro-hyoid ARCH	Great cornu of hyoid.	Thyroid cartilage (?).	Great cornu of hyoid.
3rd Crico-thyroid (?) CLEFT	*THYMUS GLAND *PHARYNGEAL DIVERTICULUM associated with double S. pyriformis Harman, full-time fœtus with left horn of thymus extending to angle of jaw (Brit. Med. Journ., 1901, ii, 1072)	*CRICO-THYROID CYSTS *Fœtal foramen *Congenital intra-laryngeal cyst (Trans. Path. Soc. Lond., xxxii, 33) ? Shattock's case (ibid., xxxiii, 289)	*THYMUS GLAND. ? Barker's case (associated with maldeveloped ear and episternal dermoid patch) (Brit. Med. Journ., 1901, i, 1204).
4th Cricoid (?) ARCH	—	Cricoid cartilage (?) (clinical evidence)	—
4th CLEFT	*THYROID GLAND, lateral portion	—	*THYROID GLAND, lateral portion.
5th ARCH	—	Hardly recognisable in man	—
	Lung (?)	Pouch of Emu (?)	Lung (?).

XX.—*Case of Acute Intestinal Obstruction due to Embolus of a Branch of Superior Mesenteric Artery, with remarks.* By W. J. Tyson, M.D., *and* W. W. Linington. *Read April* 11, 1902.

THE notes of the history of the case were kindly supplied by Mr. Clifford Hackney, with whom Dr. Tyson saw the case in consultation at Hythe on March 9, 1901.

Mrs. W., æt. 66, was first seen by Mr. Clifford Hackney on Tuesday, March 5, 1901. Her symptoms were slight pain over liver, sickness, constipation, and a little epigastric tenderness; these had been present some two or three days. Temperature was normal. On Thursday, March 7, she stated that she felt better; the bowels acted two or three times comfortably; there was less flatulence, but she was still sick, the vomit being of a greenish watery colour and free from odour. Friday, sickness became worse and abdominal distension appeared; the bowels were confined; and her general condition was much worse.

She was admitted into the hospital at Folkestone on Saturday, March 9, and was now constantly sick, the vomit being of a coffee-ground nature. There was distension of the abdomen, and constipation of over a week's duration, except on the 7th ult., when the bowels acted three times. Chest normal. Temperature 99·5°.

It was considered advisable to perform laparotomy, and this was done on the evening of her admission into the hospital, by Mr. Linington. On opening the abdomen a quantity of turbid, sanious fluid escaped; the small bowel was congested, and on tracing it down as indicated by the increased depth of colour, it was found to be slightly fixed on the right fossa. This was freed, and about a foot of fan-shaped gangrenous bowel appeared, greatly thickened, black, lustreless, and in parts sloughy; the attached mesentery was in a similar state. Recognising the condition as one of thrombosis, the affected part was excised well clear of the gangrene, and a Murphy's button used to unite the ends of the bowel, augmented by Czerny-Lembert sutures. Considerable difficulty ensued with the mesentery, owing to its

thickened and rotten state, which extended back to its spinal attachment. The dead part was removed to the extent of 22 inches, and the adjacent edges brought together on the flat, so as to give greater purchase to the sutures than in the edge-to-edge approximation. The abdomen was flushed out with hot sterile water and sewn up. Patient was very collapsed after the operation, although she improved somewhat later and was able to retain some (℥ss peptonised milk) fluid nourishment; she again became worse, notwithstanding the repeated use of stimulants and saline injections, and death occurred March 11,—thirty-six hours after the operation.

The autopsy revealed atheromatous erosion in the descending aorta, and blocking of a branch of the superior mesenteric vessel. The Murphy's button was in a good position, and the bowel water-tight; the mesentery also well secured.

The part of bowel excised was the ileum, one foot from the junction with the cæcum. The greater part of it was gangrenous, the walls of which were so greatly thickened as to gradually lessen the lumen until almost complete obstruction existed. The operation lasted just over the hour, being lengthened by the difficulty of dealing with so rotten and swollen a mesentery. It was only with great care that the ligatures could be tied tightly enough to stop the bleeding without cutting through the part included by the ligature.

Remarks.

Cases of the above character are comparatively rare, and, as far as the literature is concerned, they have been more commonly reported abroad than in England.

It seems that the general symptoms and signs are almost similar in character whether the superior or inferior mesenteric artery is involved; although certain differential points have been given, they do not seem of much value when practically considered: in both intestinal obstruction is produced. The real cause has rarely been diagnosed or even guessed at before operation or until after death.

The first prominent symptom is abdominal pain, closely resembling an ordinary attack of colic. Nausea is frequent, and comes on fairly early. Vomiting follows, with a gradual change in the character of the vomit. Constipation is the rule, but diarrhœa has been commonly reported. Tension and tympanitic swelling of the abdomen are late symptoms, indicating the advent of peritonitis. Extreme fœtor of the

stools, should there be diarrhœa, is of serious omen, and points to gangrene of the bowel having taken place. Profuse intestinal hæmorrhage has also been recorded, and when meeting with this symptom it is as well to bear in mind that embolus of the mesenteric artery may be the cause.

Prognosis is always most grave, but not absolutely fatal; of course this must be better if only one branch rather than several are involved. It is doubtful, when the main vessel has been occluded, whether recovery has ever taken place.

The greater the arterial obstruction the more severe the symptoms. As regards the source of the embolus, the left heart seems to be the most common one, and after this the abdominal aorta, as in the present case. There have been a few cases of thrombosis of the superior mesenteric vein recorded giving rise to intestinal obstruction, the artery not being affected, notably Fagge's and Bradford's cases. Where the swelling of bowel from infarcts has occurred, the tumour has been mistaken for one of malignant nature.

Treatment.—Early abdominal section gives the only possible chance; we have not been able to find any recorded instance of recovery after delayed operation, and it is quite conceivable, had the foregoing case been operated on a few days earlier, the patient might have recovered. It is quite evident that kneading the abdomen in such cases must be absolutely wrong, and also that opium in other than small doses would mask and obscure the real cause of the disease.

XXI.—*Cerebellar Abscess with Sudden Paralysis of the Respiratory Centres.* By ARTHUR E. BARKER. *Read April* 11, 1902.

THE special point in this case, which I think deserves to be recorded, is the clear evidence of sudden paralysis of the respiratory centres, due to encroachment of the abscess upon the floor of the fourth ventricle, while the nervous mechanism of the heart remained intact for two hours and a half under artificial respiration. Even then the heart was beating strongly, and only ceased when the artificial respiration was discontinued.

Beatrice H., æt. 14, had been quite well until three months before admission into University College Hospital on December 14, 1901. She then suffered from scarlet fever and right otitis media purulenta, and was treated for it at another hospital. A fortnight before admission the patient began to suffer severely from earache in the same ear, with intermission of the discharge. Poultices were applied for this, with relief on return of the discharge. On Sunday, December 8, the patient began to suffer from extreme weakness, the slightest movement requiring great effort. At this time the earache became more severe, and the patient vomited. Since then she has been in bed, and has taken very little food. During the week severe headache, especially on the right side and behind, was complained of. On Wednesday, December 11, the pain became very severe, and a prolonged shivering fit took place. Since then until admission on the 14th there has been a similar rigor every day about noon, four in all. She became drowsy early in the week, and during the last three or four days has taken no notice of the surroundings, and only occasionally complaining of headache. On the day of admission (Saturday, December 14) she vomited once in the morning, and for the last twenty-four hours there had been some cough.

On admission she was seen to be pale and wasted, and lay quietly in bed, either on the back or side, in the latter case with the head much retracted and the limbs much flexed. There was some restlessness from time to time, with moaning and carrying the hand to the head. The patient seemed to understand what was said to her, but only answered "yes" or "no" to questions. She

could put out her tongue and move all her limbs freely. There was a profuse thick and intensely fœtid discharge from the right ear. Examination showed much swelling of the meatus, preventing the deeper parts from being seen. The left membrana tympani appeared to be normal. There was no swelling over the mastoid process, but there was well-marked tenderness there. The headache appeared to be chiefly in the occipital region. The pupils were normal and reacted well to light, and the optic discs showed no morbid change. There were no signs of ocular or other paralysis. The patient swallowed well. On the side of the neck, reaching from the angle of the jaw downwards to the larynx, there was well-defined tenderness and some induration along the line of the deep jugular vein. This appeared to mask the pulsation of the carotid, which was much feebler than on the left side. There was no cough now, or any sign of lung disease. There was no paralysis of the limbs, but the grasp of the right hand was distinctly weaker than that of the left. Right knee-jerk much exaggerated; left slightly so. Plantar reflex well marked. Extensive response of great toe on left; slight on right. No interference with sensations. Pulse feeble, 66; temperature 99°.

On December 15, at 10.30 a.m., I again examined the patient. There had been no change since the evening before, except that the temperature in the night had been 98°. No vomiting or rigor had occurred, and the bowels had been well opened. The pulse was 66 per minute and the respirations quite normal. She was still semi-conscious, and answered questions.

I had now no doubt that the patient was suffering from an intra-cranial abscess, with thrombosis of the lateral sinus and deep jugular vein. I based the diagnosis upon the history of chronic otitis media purulenta of at least three months' duration, with an exacerbation fourteen days before admission. This, and not acute attacks, is the history of nine tenths of the cases of intra-cranial abscess which are met with. I learned this while in charge of the ear department for many years, and have often since been surprised at hearing a diagnosis of cerebral abscess made in recent acute cases of otitis media purulenta, or even serosa, which in my experience practically never lead to such abscesses. I have good reason to know from what I have seen in hospital and private that this fact is not sufficiently widely known.

In addition, we had in this case clear evidence of thrombosis of the lateral sinus with extension of the process into the deep jugular vein. This was indicated by swelling, induration, and great tenderness over the vein at the angle of the jaw. But in spite of this septic phlebitis the temperature was practically normal or subnormal instead of elevated. This character of the temperature we have learned is one of the most significant signs of a well-matured abscess. Then there had been successive rigors each day, and the patient had vomited several times. There were no convulsive movements and no paresis, nor was there any sign of optic neuritis at all compatible with the diagnosis of intra-cranial abscess. I therefore had every preparation made to operate on the brain or cerebellum at 3 p.m.

Immediately before the patient was lifted from her bed to be brought into the anæsthesia room, at 2.50 p.m., Sunday, December 15, 1901, she looked in the same condition as when I saw her in the morning at 10.15. She was semi-conscious; colour good; temperature 98°; pulse 60. She arrived in the anæsthesia room at 2.55 p.m., when I was at once called from the operating theatre to see her, as she was livid and not breathing. No anæsthetic had been administered. I found her quite blue and absolutely without respiratory movements. The heart was beating strongly and rapidly. Artificial respiration was at once commenced, and in a minute or so the normal colour returned to the skin, aided by oxygen, which was also administered. The air entered the lungs freely. Still there were no signs of normal respiration. The pupils were dilated and did not react to light, and the conjunctival reflexes were absent. Respiration was kept up for half an hour, but without avail. At the end of this time the hand pulse was strong at 128. It appeared clear then that an abscess had burst and was inhibiting the respiratory centres. I then sat the child upright, with her legs hanging over the edge of the table, thinking that perhaps cerebral pressure might be relieved thereby and the centres might recover. All those present thought that at this moment there were a couple of slight sighs, but perhaps this was only due to the descent of the diaphragm. As she was losing colour again, artificial respiration was recommenced and carried on steadily. At the end of an hour there was no natural breathing, while the heart was beating strongly now at 120. Half an hour later the colour was still good under the artificial breathing, and the pulse was 100 in the heart and larger arteries. It could not

be felt in the wrist and foot. The temperature in the rectum was 95·8°. I then thought that perhaps the opening of the cerebral abscess might relieve the pressure, and rapidly, through a small incision, opened the skull with a ¼-inch trephine over the temporo-sphenoidal lobe. No pus was found. I passed the needle into the posterior corner of the lateral ventricle, and immediately evacuated a large quantity of almost clear fluid under considerable pressure. This slowed the heart a little. I then similarly opened over the lateral front of the cerebellum, and after a few punctures reached foul gas and pus anteriorly, which filled a ⅔ test-tube about two inches full. This, however, had no effect on the respirations or heart. After tying some small bleeding vessels I dressed the wound, and gave directions that artificial respiration should be kept up until the patient was obviously dead. This was done for another three quarters of an hour, when the eyeballs seemed quite flaccid and there was no response to artificial respiration, and it was discontinued. But the heart was still beating as before, two hours and thirty-five minutes after natural breathing had ceased.

At the *post-mortem* examination a large cerebellar abscess was found in the usual situation anteriorly and internally, which had been partially emptied by the operation. There was no general meningitis, but there was a little lymph on the surface of the cerebellum corresponding to the abscess as it lay against the petrous bone. On looking into the fourth ventricle there was no evidence of actual pus in its fluid, but on the floor a portion of the abscess was seen which was about to burst if it had not actually done so. On being touched with a probe, pus welled out at one point into the ventricle. The general cavity of the abscess was about the size of a walnut. The lateral ventricles contained a quantity of slightly turbid fluid; otherwise the brain was normal. The lateral sinus was thrombosed with purulent clot, which extended into the deep jugular vein for about two inches. The lungs and heart were normal except for a number of petechiæ in the former on the left side. The punctures in the brain and cerebellum were well placed, and showed nothing unusual. One, as described, had reached the lateral ventricle.

It is not a common experience to have such clear evidence of direct interference with the respiratory centres as in this case, while all the other centres are almost or altogether unaffected. I have therefore thought it worthy of being recorded, especially as it gives a good illustration of the long

time for which the heart can continue to beat after natural respiration has ceased. Here it was over two and a half hours, and then it only gradually stopped when artificial respiration was abandoned. I have only been concerned in one case where anything parallel was observed. This was a case I was asked to see by Dr. Bradford some years ago, in which a small aneurysm at the base of the brain had burst, and the blood pressed upon the floor of the fourth ventricle. Here the heart continued to beat for four hours after respiration had ceased, as I am informed by Dr. Bradford.

The whole cerebellum, on removal, was hardened and then cut in successive horizontal slices from above downwards by Mr. Trotter, our surgical registrar. I have them mounted, and now present them for examination. The extent and position of the abscess is here particularly well seen. It must have been very large and tense before evacuation. Even now, in its more or less collapsed condition, it is very extensive, occupying most of the right lobe of the cerebellum. But the most interesting feature in it is the direct prolongation of one portion of it inwards until it presses on the lateral ventricle, if not actually bursts into it, of which rupture Mr. Trotter is inclined to think there is no absolute evidence. But in the fresh state, as I have stated above, a touch with a probe caused a welling out of pus into the ventricle. Be this as it may, the abscess was close enough to the floor of the fourth ventricle to produce pressure effects upon the respiratory centres. The only difficult point to explain is the absolutely sudden onset of the respiratory trouble.

XXII.—*The Simulation of Acute Peritonitis by Pleuro-pneumonic Diseases.* By H. L. Barnard, M.S. *Read April* 11, 1902.

Introduction.—Thoracic diseases in some cases produce the most deceptive abdominal signs. Modern standard works of medicine contain only a meagre reference to this condition, and it is apparently unknown to surgeons, if we may judge from their text-books. The great development of abdominal surgery in recent years has made the recognition of this simulation a matter of practical importance, for a mistaken diagnosis is frequently followed by an unnecessary and dangerous operation.

Case 1. *Right diaphragmatic pleurisy and pneumonia, with marked abdominal signs, simulating appendicitis with peritonitis.*—This case I owe to the kindness of Dr. Kidd. A little girl, Eliza H., æt. 11, was admitted to the medical wards of the London Hospital in 1895, complaining of pain in the head, but especially in the abdomen. Three days before admission she had severe pain down the right side of the abdomen, and vomited bilious fluid several times. When she was first seen at the hospital her abdomen was rigid, and especially tender in the right iliac fossa. Her bowels had not been open for three days, and her tongue was brown and dry. Her pulse was 104, full, and not easily compressed; and her temperature 103° F. The thoracic signs, which at no time were very marked, were almost absent on admission; she was, however, breathing about 40 to the minute, and cut her respirations short, because they hurt her, in a manner characteristic of pleurisy. She had, moreover, tenderness along the attachment of the diaphragm, and the veins were dilated there.

By the fourth day after admission there were obscure signs at the right base of some pneumonia, breath-sounds a little diminished, and a few moist râles, but these were also heard at the left base. Her temperature, which had remained about 103°, fell by crisis to normal on the fourth day,

but soon became irregular, and for four or five weeks frequently reached 102°. During this time the signs of pneumonia and diaphragmatic pleurisy became more distinct, and at last left no doubt as to her condition; but had she been admitted to a surgical instead of a medical ward, and at the present time when so many cases of appendicitis are submitted to early operation, instead of six years ago, I cannot help thinking that her abdomen would probably have been explored.

Case 2. *Pleuro-pneumonia of right base; acute abdominal pain and vomiting; no lung signs; herpes labialis; empyema; recovery.*—This case I owe to the kindness of Mr. Dean and Dr. G. Schorstein. A little boy, John E., æt. 10, was admitted to the London Hospital in 1899, as acute peritonitis after an injury. Two days before admission he had received a blow in the lower part of the chest or upper part of the abdomen. On the following day, the day before admission, he was seized with severe pain in the abdomen, chiefly in the upper part, and vomited twice.

On admission his belly was somewhat distended, tender, and rigid all over, but especially so in the epigastric region. His bowels had been constipated since the pain commenced, and an enema only gave a small result. He lay with both legs drawn up. His temperature was 99·5° F. on admission, but rose to 103° F. the same evening. His pulse was 130 to 140, but of good volume and strength. His respirations were 44 per minute and catchy. The examination of the thorax gave absolutely negative results.

Soon after admission he developed herpes labialis, and was transferred next day to the Medical side. No signs had yet appeared in the thorax, but on the following day there was undoubted consolidation at the right base. The temperature fell by crisis on the ninth day of the disease, but seven days later it rose again and became irregular, and an empyema developed on the right side. This was opened and drained, and he then made a good recovery.

Case 3. *Double basal pneumonia, with right diaphragmatic pleurisy, in a girl, the subject of a gastric ulcer; acute epigastric pain, with collapse; laparotomy; death.*—This case is certainly the most perplexing one I have yet heard of. I am indebted for it to Mr. Eve. A girl, Nellie F., æt. 17, had been attending the Out-patients' Department of the London

Hospital for some months for marked anæmia and gastric ulcer. She had had a similar attack to the one I am about to describe once before. One morning in 1899 she started for work as usual, and on her way was seized in the street with violent epigastric pain and vomiting. She was brought to the hospital at once in a state of collapse. Her pulse was 120 and very thready; her temperature 104·5° F. Her abdomen was rigid, motionless, very tender, and distended, and these signs were most marked in the epigastric region.

She was admitted to the surgical wards as a case of ruptured gastric ulcer, and within two hours Mr. Eve opened her abdomen. He explored the anterior and posterior surfaces of the stomach, the greater and lesser curvatures, but found no trace of gastric ulcer, nor was there any peritonitis. Unfortunately ether was the anæsthetic given. Misfortunes followed fast. Her cough became so violent that a coil of intestine and some omentum escaped between the stitches and had to be washed and returned, and the abdomen again sewn up.

On the second day after admission it was clear that she had right basal pneumonia, but her temperature had fallen to 101°. On the third day it rose again to 104°, and signs of consolidation appeared at the left base, and she died on the fifth day from the commencement of the attack.

At the *post-mortem* double basal pneumonia and right diaphragmatic pleurisy were found, and in the stomach a shallow ulcer the size of a sixpence which was not even near perforation. There was no peritonitis. I think all will admit that this was an impossible case to diagnose correctly. Looking back at the case, the only point which should have aroused suspicion was the temperature, 104·5°. No record of the frequency of respiration was kept.

Case 4. *A case of pyo-pneumothorax of the left base simulating appendicular peritonitis or left subphrenic abscess of gastric origin.*—A Hebrew, Samuel B., æt. 34, was admitted to the London Hospital under a medical colleague for "colic and ? peritonitis" in 1901. I was asked to give a surgical opinion on his abdominal condition. He had not been well for a year, suffering chiefly from pain and wind in the stomach after food, but without vomiting. For several weeks he had also pain in the left lower and back part of his chest, with a bad cough and night sweats and wasting. He had been constipated for twelve days.

When I saw him his temperature was 101° F.; his pulse about 100, and fairly strong; his respiration 38, and his nares vigorously dilated at each inspiration. He was a little icteric, but not really jaundiced. His abdomen was rigid and very tender all over, but I could make out no local dulness or resistance, and the abdominal wall relaxed between each respiration. The base of the left lung behind was dull on percussion. Vocal resonance and tactile vocal fremitus were diminished. Such breath-sounds as could be heard were of a somewhat tubular character. The house physician thought the right iliac fossa more rigid and resistent than the rest of the abdomen. The medical registrar was of opinion that the signs indicated left subphrenic abscess due to a perforated gastric ulcer, but the stomach resonance was normal, and the spleen was not displaced. My medical colleague agreed with me that the signs appeared to indicate trouble above the diaphragm, and arising from the lower lobe of the left lung.

Ten days later it was clear that he had pyo-pneumothorax at the left base, probably arising from a tubercular focus in the apex of his left lower lobe. A rib was accordingly resected, and his temperature fell to normal, he put on flesh, and lost all pain; but a sinus persisted when he left the hospital.

CASE 5. *A case of pyo-pneumothorax of the right base simulating appendicular peritonitis.*—On September 12, 1901, I was called late one evening to see a man in the wards of the London Hospital said by my house surgeon to be suffering from "appendicitis and general peritonitis."

Two days before admission the patient was running for a train on his way to work in the morning when he was seized with severe pain in the right iliac fossa. He gave up work at 1 p.m. on the same day and took to his bed. Next day his bowels were well opened by a pill and castor oil. He had no vomiting. He had never been seriously ill before. When I saw him in the wards it was clear that he was seriously ill and in great pain. His temperature was 100° F.; his pulse 80; his respiration 43; and his tongue furred and dry. His abdomen was hard, rigid, and tender all over, but perhaps a little more so in the right iliac fossa. It relaxed, however, between the respirations for a second and became soft, and there was no marked distension or localised dulness or resistance. The heart was normal, and the examination of

the lungs showed nothing but a little bronchitis and emphysema. I refused to operate, because whilst the abdominal signs appeared to me at least doubtful, the respiration (43 a minute) with a pulse of only 80 appeared to indicate that the lungs were the source of the trouble. The thing, however, which really decided me not to operate was that I did not think he could safely take an anæsthetic. I carefully examined him again next day with a medical colleague. The abdominal signs had disappeared almost entirely, and dulness at the right base with diminished breath-sounds and tactile vocal fremitus, the presence of a pleural rub, and the absence of tubular breathing left no doubt that he had right basal pleurisy. Eight days later the signs at the right base had much increased, and pus was found by an exploring needle. A rib was resected, and 24 oz. of pus with air were evacuated. The condition was then found to be one of pyo-pneumothorax.

I saw him once again about a couple of months ago. He had been attending the out-patients' for a sinus left by the empyema. An energetic dresser had syringed out his cavity with some antiseptic, and had nearly choked him by the passage of the fluid through the orifice in the lung into the trachea.

Case 6. *Traumatic left pleurisy, with abdominal signs; laparotomy.*—A labourer, æt. 38, was admitted under my care in February, 1901, giving the following history:—He was assisting to unload deal planks from a barge when one swung round suddenly whilst hanging from the crane and struck him violently over the lower left ribs, precipitating him twelve feet into the barge, where he alighted on the back of his head. He was slightly concussed, and had a scalp wound in the occipital region. He soon recovered from the shock of the fall, and the only sign of the injury which could be made out was a fracture of two of his left lower ribs, probably the ninth and tenth; but he was so muscular a man, and the part so tender, that the exact ribs broken could not be determined. Within twelve hours of admission a pleuritic rub was heard in the region of the broken ribs.

Next day acute abdominal signs appeared. He complained of violent abdominal pain, which was worse down the left side of the abdomen. His belly was rigid all over, but especially in the left hypochondrium and lumbar regions, but the abdominal wall still moved on respiration. Ill-defined dulness was found down the left side, and this was thought to

be due to blood in the peritoneum. He vomited continuously, and the vomitus soon became bilious. His bowels had not been opened since he was admitted, and an enema of a pint and a half of soap and water was given. This was retained. Retention of urine, which had been present since admission, persisted. His temperature was 99°, and his pulse 75 and of good volume. A diagnosis was made of laceration of the spleen, with recurrent hæmorrhage; and laparotomy was decided upon.

I must admit that this diagnosis was largely due to the fact that within a fortnight a very similar case had been admitted to the same ward and did not develop any marked abdominal signs until the fifth day, when he was to have left the hospital. On that day he collapsed, and his abdomen became distended, and much free fluid was discovered. Laparotomy was performed, and the spleen found torn nearly in two. He died of hæmorrhage on the table whilst the clots were being displaced to find the pedicle.

The abdomen of the present case was opened in the left linea semilunaris, and the abdominal contents found absolutely normal. The dulness was explained by the enormous muscular development of the abdominal wall, which was so great that the recti and lateral abdominal muscles projected into the peritoneum as rounded masses, which were at first thought to be hæmatomata. The man was by trade a deal-runner. Some idea of the spasm may be gained by the fact that strand after strand of ordinary silk-worm gut snapped without drawing the edges of the incision together, and finally the stoutest silk had to be employed and the anæsthetic vigorously pushed before the abdominal wound was closed. He healed by first intention, and made an uninterrupted recovery.

The Simulation of Acute Peritonitis by Pleuro-pneumonic Diseases.

Andral (1) appears to have recognised this condition clearly in 1836, and in his book narrates several typical cases.

Watson (2), in his admirable text-book of Medicine, quotes Andral, and refers to cases of his own in which pleurisy at the right base simulated hepatitis, but he does not appear to have recognised the possibility of confusing diaphragmatic pleurisy with acute peritonitis. Fagge knew the condition

well; in his text-book he says, "In exceptional cases the pain is referred to the terminal branches of the intercostal nerves; to the hypochondrium, so as to lead to a mistaken diagnosis of hepatitis; to the loins, so that the case has been called one of lumbago; or to the neighbourhood of the umbilicus, so that peritonitis has been suspected;" and in a note he adds, "I have myself had a patient whose sole complaint was of pain in the crista ilii. I feel sure that if it had not happened that a short time before my attention had been specially directed to this question I should have failed to discover that he had pleurisy, although on applying my stethoscope I at once heard a rub.—C. H. F." (3).

Modern medical writers for the most part are vague in their reference to this class of case, and many do not even refer to them at all. The condition is altogether unknown to surgeons if we judge from their text-books, and this is all the more unfortunate, as it is they who will open the belly of the pleuritic patient should they be ignorant of this condition after they have caused ether to be administered. My excuse for this paper is to call attention to a pitfall in surgery into which, as you have heard, I have fallen myself.

The thoracic diseases which can give rise to such severe abdominal signs as to simulate peritonitis are all situated in the lower part of the chest, either in the base of the lung or the adjacent pleura, diaphragmatic or parietal. Three of my cases were *pleuro-pneumonias* of the base of the lung and involving *the diaphragm.* Two were cases in which apparently a tubercular focus in the apex of the lower lobe ruptured into the pleural cavity, producing *pyo-pneumothorax.* One case was most interesting in that the *pleurisy* was *traumatic* and due to a localised injury, and associated with broken ribs; it was recognised, and yet the abdomen was opened, so definite were the abdominal signs.

The abdominal signs and symptoms which may arise in such conditions are conveniently divided into (*a*) *parietal* and (*b*) *visceral.*

(*a*) *Parietal.*—The abdominal wall is *acutely tender and painful.* The tenderness often extends over the whole belly, but in some cases it is unilateral; it may be present only above or below, or be more intense in these situations. It will readily be seen that such acute abdominal pain and hyperæsthesia in the right hypochondrium may be mistaken for hepatic and biliary diseases, whilst in the right iliac region an appendicitis would at once suggest itself, and

in the epigastrium a perforated gastric ulcer. The abdominal muscles are, moreover, in *spasm*, so that the belly wall is board-like and hard. So great is this spasm that the deepest anæsthesia is necessary to relax it, and the pupils will be widely dilated before the belly becomes soft. This has practical application, for if the intestines are allowed to escape they will be returned to the belly with the greatest difficulty, and the surgeon will find that the stoutest silk alone will stand the strain necessary to close the wound. *The respiration is of the costal type*, but it will be found on placing the hand upon the belly wall that this relaxes for a moment at each inspiration, and this is an important diagnostic sign. Writers on diaphragmatic pleurisy insist on the importance of unilateral exaggeration of costal respiration on the side of the chest affected.

(*b*) *The visceral signs* are distributed along the whole length of the alimentary canal. *Gastric disturbance* leads to nausea, hiccough, and vomiting, which may be urgent and become bilious. *The intestines* are apparently more or less paralysed, so that the abdomen becomes distended, and this, with the tenderness and spasm of the abdominal wall already referred to, produces a most deceptive resemblance to peritonitis. Constipation is nearly always present, and enemas may be retained, so that, apparently, *the rectum* has lost its irritability. Even the profound *vascular changes* we are in the habit of associating with severe lesions in the splanchnic area are frequently present, at least at the onset of the attack, although it must be admitted they do not often persist or become more severe as the disease develops.

The patient is often collapsed at the onset of the pain. The extremities are cold, and beads of sweat may break out on the forehead, whilst the features are pale and pinched; but in none of my cases did I see the rings round the eyes, or the dry, shrivelled tongue one associates with general suppurative peritonitis.

The diagnosis is nearly always easy if suspicion is aroused and the surgeon is aware of this close simulation. The chief points to be observed are the rapidity of the respiration, often 40 a minute or more, which is out of all proportion to the pulse rate, which is seldom so frequent as 100 a minute, and is not weak or wiry. The respiration will often exhibit the curious catch at the top of inspiration which is characteristic of pleurisy. The abdominal tenderness is, moreover, found to be chiefly superficial, and firm, deep pressure with the flat

hand may be permitted. It will then be found that the abdominal wall becomes soft for a moment at each inspiration, which is not the case in peritonitis. The absence of rings round the eyes, and a temperature of about 103°, should also make one hesitate to diagnose a serious and general peritoneal infection.

When once a suspicion has dawned on the mind of the surgeon, the patient is comparatively safe, for an examination of the suspected side of the thorax will reveal some dulness or friction-sound, or some excess of costal movement on that side over the other which will lead him to pause and perhaps consult his medical colleague; but it must be frankly admitted that direct thoracic signs are often almost entirely lacking for twenty-four hours or more. The great danger after all appears to arise from auto-suggestion by the patient's surroundings and the circumstances under which he is first seen. The surgeon is summoned by his house surgeon perhaps in the dead of night to see a case of "acute peritonitis, probably of appendix or gastric-ulcer origin." He goes to his well-known ward where he has seen series of cases of every phase of abdominal disease, but seldom or never pleurisy or pneumonia. There he finds a patient suffering from acute abdominal pain, perhaps vomiting, and the house surgeon tells him he has retained an enema. The pain the patient is in, and perhaps his cries when he is being examined, disturbing the whole ward as they do at night, prevent a complete examination. It may be that the surgeon is not quite satisfied that the signs undoubtedly indicate general peritonitis, but his clinical experience at once reminds him how vague the signs of general peritonitis may be; of the case of ruptured liver or spleen in which the signs were so vague for three or four days that operation was postponed until too late; or of that case of ruptured gut which, for two days, was supposed to be abdominal contusions. Under such circumstances it is really not surprising that patients exhibiting the signs I have mentioned are sometimes operated on. This is an accident always to be deplored, for very frequently ether is administered, and adds bronchitis to the pre-existing lung condition, whilst an extensive abdominal incision seriously hinders coughing. The only guard against such deplorable accidents is to recognise the possibility of their occurrence; and that is my excuse for this paper and the record of these cases.

The pathology is one of referred pain, and divides itself into three heads:

1. *Irritation in continuity* of the lower six dorsal nerves will explain the hyperæsthesia and spasm of the abdominal wall. These intercostal nerves supply the diaphragm and lower part of the pleura, and here their branches, if not their trunks, will be irritated by acute inflammatory processes. In this connection it is interesting to note that three of my six cases terminated in suppuration. This seems to indicate that the inflammation, in order to produce such abdominal signs, must be severe enough to penetrate to the nerve-trunks themselves and produce actual neuritis. The terminations of the seventh to the eleventh dorsal nerves pass to the abdominal wall, and supply the skin and muscles there. If the seventh, eighth, and ninth nerves are chiefly affected the acute epigastric tenderness and spasm will closely simulate a perforated gastric ulcer; or if chiefly hypochondriac, and on the right side, hepatitis or gall-bladder trouble. When the tenth dorsal nerve is mainly affected, the pain is referred to the umbilicus, as it is in so many abdominal complaints, whilst the area of the tenth and eleventh dorsal nerves on the right side is that in which the signs of an acute appendicitis develop. Moreover, the presence of tenderness and spasm on the left side in the tenth and eleventh dorsal areas does not exclude appendicitis, for in the early stage of this affection the pain may be referred to that side.

2. *Referred visceral pain* has been most exhaustively studied by my colleague, Dr. Henry Head (4), and it explains referred abdominal signs from lung diseases without pleurisy. The seventh, eighth, and ninth dorsal rami communicantes are those which supply the lungs. When broncho-pneumonia, phthisis, or bronchitis, but not lobar pneumonia, supervene, even without pleurisy, impulses may pass up to the posterior root-ganglia of these nerves and there produce such changes that normal impulses passing through these ganglia from the terminal branches of the nerves in the epigastrium and upper abdominal wall are exalted into painful impressions. The spasm follows in a reflex manner from the pain. Dr. Head tells me he has seen such cases, but none of those related here are explicable on this pathology.

3. *Of the pathology of the visceral signs* to which we have referred, gastric, intestinal, rectal, and vascular, we know very little. Perhaps they are the result of a reflex in the reverse direction to that to which we have just alluded, so

that irritation of the six lower intercostal nerves by a pleurisy produces disturbance in the distribution of the three splanchnic nerves which are derived from the same level of the spinal cord. On the other hand, it may be due to irritation of these nerves in continuity as they lie beneath the pleura or pass through the diaphragm to the great sympathetic plexuses and ganglia of the abdomen.

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XXIII.—*Congenital Diffuse Lymphangioma terminating in Lympho-sarcoma after Lymphangitis, with a continuous Hectic Fever for three months affecting the Lower Extremity of a girl aged four.* By Walter G. Spencer, M.S. *Read May* 9, 1902.

A GIRL æt. 4, admitted to the Westminster Hospital on April 5, died on July 5, 1901.

Clinical history.—The father stated that the child had been born with the right leg larger than the left, but had always run about and played like other children, so that no attention had been paid to it.

A few days before admission the limb had become much swollen and inflamed without any assignable cause.

On admission the whole right lower limb was much enlarged, the foot, lower leg, and upper thigh slightly so, by œdema; the upper third of the leg and lower third of the thigh were more than double the opposite side, red, tensely elastic, and very tender. The temperature was high, reaching 105° F., the pulse rapid and weak. Œdematous fluid drawn from the limb contained at one time streptococci, at another staphylococci, but there was no suppuration discovered, nor other affection.

The patient was at first given repeated 5-c.mm. doses of antistreptococcal serum without any effect whatever. As the child seemed very ill amputation through the middle of the thigh was proposed, but declined by the father. In view of the termination of the case it is doubtful whether amputation would have been of service. When the father later on desired amputation it was clearly too late, for then the new growth had become evident in the iliac fossa.

To relieve pain and tension small incisions were at various times made into the thigh, upper leg, groin, and loin, and boric acid fomentations employed. From the incisions escaped serous fluid with fibrinous casts of the distended lymphatic space—white globular masses the size of peas and beans. As the limb was kept elevated the lower thigh tended to become less swollen, the upper thigh and buttock to enlarge, and the groin and loin to get more œdematous. After six weeks the right labium majus had become so large and tense

as to require puncture. After ten weeks the right loin was tense with œdema, and from small incisions the casts of the lymphatic spaces already mentioned escaped. Meanwhile the œdema of the abdominal wall, in the inguinal region, became replaced by hard masses filling the iliac fossa. For the first time, just before death, a collection of pus formed subcutaneously in the upper part of the thigh; no chylous fluid ever escaped, nor did lymph vessels appear on the skin, which was not even rugose.

The patient suffered from pain to a variable degree; vomiting occurred nearly every day; there was sweating when the temperature rose high, but there was never a rigor.

The temperature showed that there was every evening a rise to 103° or 105° F., and an early morning fall to normal or below, to 97° or even 96° F. The pulse continued throughout about 130 to 140, but on many days rose higher, to 170 or even 180. The respiration varied between 35 and 40 per minute.

Post-mortem examination.—There was extreme emaciation of the entire body, except the right lower limb, the labium majus, and the iliac fossa. Beyond a little broncho-pneumonia, all other organs were normal. The right lower limb was much enlarged; the circumference at the middle of the right thigh was 12½ inches against 6 inches on the left, and there was a corresponding disparity in other parts. In the neighbourhood of the right sacro-iliac synchondrosis, and extending thence into the substance of the psoas and iliacus muscles and into the pelvis, was an ill-defined spongy mass, which on section showed definite angiomatous cavities, partly filled by clear lymph which escaped on section, partly by blood-clot. No definitely enlarged lymphatics were seen. The tissues of the affected limb were enormously œdematous down to and including the periosteum, and in the subcutaneous tissue were numerous cysts surrounded by lymphangiomatous tissue. But the skin, except for the surgical incisions, was not altered.

The Peculiarities presented by the Case.

It was of congenital origin, as distinguished from acquired. The disease consisted in a dilatation of lymphatic spaces with a new formation of intercystic tissue replacing connective tissue, subcutaneous, intermuscular, and periosteal, whilst there was no sign of lymphatic vessels dilated by obstruction

above. The skin was not involved, no vesicles formed on the surface, so that no lymph nor chyle escaped.

The case is thus to be distinguished from that fully described by Day, including the termination and post-mortem examination (in the *Clinical Society's Transactions*, 1869, vol. ii, p. 104, and 1878, vol. xi, p. 214). A boy was first noted, when aged two and a half years, to have the right leg larger than the left, which slowly increased with intermittent attacks of inflammation and extended to the prepuce. Chyle began to escape at five years of age, and the patient's history until his death from exhaustion, when aged sixteen, was that of intermittent discharges of chyle and inflammatory attacks. Post-mortem examination revealed only an involvement of the subcutaneous tissue, not of the deeper connective tissue, nor of the lymphatic glands, nor of the lymphatic trunks. There was no new growth.

A nearer parallel is to be found in the case described by Sir Thomas Smith as "nævoid elephantiasis" (*St. Bartholomew's Hospital Reports*, 1869, vol. v, p. 147).

In a girl the left limb had been enormously enlarged at birth, and at nine months the enlargement involved also the buttock. Not only was the skin affected by a wide-spread nævus, but the whole limb was involved by a venous or cavernous hæmangioma.

The case now described was a lymphangioma not connected with the blood-vessels further than by the extravasation of blood into the lymphatic spaces. A full post-mortem examination was not made in Sir Thomas Smith's case, but there was no evidence of new growth further than of the venous hæmangioma.

A case described by Langhans (*Virchow's Archiv*, 1879, Bd. 75, S. 293), of lymphangioma affecting the left leg in a boy aged seven months, is similar to mine in that the subcutaneous intermuscular, perivascular, and periosteal connective tissues were all involved in a degree most marked from below upwards whilst the skin was free.

Langhans especially describes the disease as a diffuse lymphangioma, consisting not only of a dilatation of lymphatic spaces, but of a new growth in the connective tissue. This child died from pyæmia at seven months, following excisions of small pieces from the leg, the disease not having extended beyond the thigh, and no solid new growth being found, although, had the child lived, the course pursued might have been similar to mine.

Cases of lympho-sarcoma have arisen in the lymphatics of the groin of children, *e. g.* in Hobson's case (*Pathological Society's Transactions*, 1882, vol. xxxiii, p. 300). A large mass involved the right inguinal region and pelvis of a girl aged four with extensions in the liver, pancreas, kidneys, ovaries, etc., but this had not been preceded by a diffuse lymphangioma of the lower limb.

There appears a close analogy between the disease in my case and that of some forms of macroglossia, except that there was no lymphatic nævus on the skin.

I may be allowed to refer to the chapter on macroglossia in *Diseases of the Tongue*, by Butlin and Spencer, p. 305, also fig. 21, p. 267. There are mentioned instances of congenital lymphangioma of the tongue ending in lympho-sarcoma. One described by Hutchinson was noted at ten years of age to have a rough papillary growth at the base of the tongue, and this slowly grew to a large mass requiring the removal of the tongue; two years later a recurrent growth sprang up in the mouth and caused death by hæmorrhage. Eve has described a Hunterian specimen in the College of Surgeons' Museum in which a lympho-sarcoma had grown on a lymphangioma. A boy described by Perkins had at four years a lymphangioma the size of a pin's head, at twelve years it had grown to the size of a walnut, at twenty-five it had filled the mouth and involved the submaxillary glands, and the growth recurred after the removal of the anterior three fourths of the tongue. In our description of macroglossia we show that masses of small round-cells are met with between the cysts in quite early cases of a similar appearance microscopically to lymphadenoma and lympho-sarcoma.

DESCRIPTION OF PLATE X.

To illustrate Mr. Spencer's paper on Congenital Diffuse Lymphangioma terminating in Lympho-sarcoma.

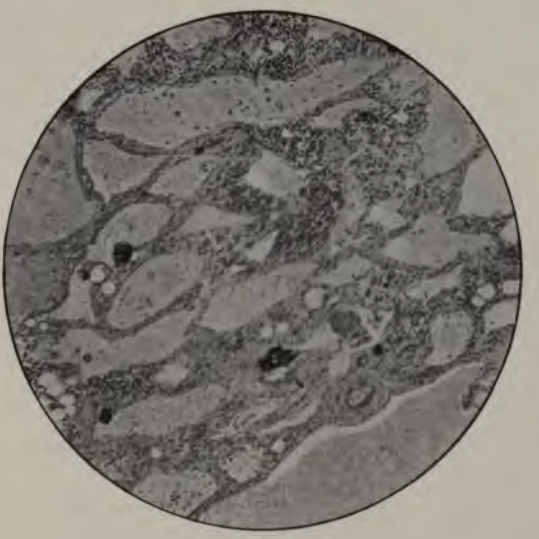

FIG. 1.

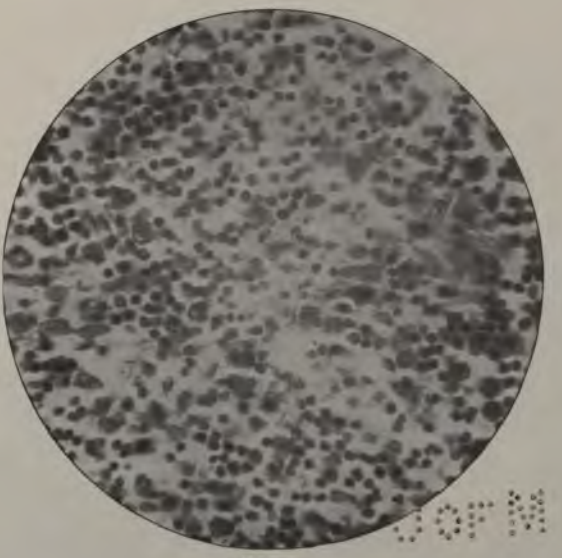

FIG. 2.

Bale and Danielsson, Ltd.

XXIV.—*A case of Primary Sarcoma of the Synovial Membrane of the Ankle-joint.* By G. R. Turner. *Read May* 9, 1902.

ALBERT K—, æt. 28, was admitted into St. George's Hospital in January, 1902. Two months previously he had knocked his foot against a brass screw, and some three weeks after this injury he first noticed a swelling in front of the right ankle. The tumour had slowly increased in size, but beyond making his foot "seem weak" caused him little inconvenience. There was no history of tubercle, and he had had no previous illness of any importance. In front of his right ankle, just anterior to the external malleolus, was a hard roundish swelling about $1\frac{3}{4}$ by $1\frac{1}{2}$ inches in diameter, apparently not elastic or fluctuating, and which could not be reduced into the joint. There was no obvious connection with the tendons. A cutaneous nerve was stretched tightly over the tumour.

On January 16 it was removed, and was found to be a growth springing from the synovial membrane of the ankle-joint. On incising the anterior ligament the tumour came away very readily. It was moulded to the astragalus on its joint surface, which was concave and glistening. There were outgrowths and excrescences in front and on the edges, and in places it was of a dark brown colour.

Dr. Rolleston reported as follows:

"Microscopically the growth was a mixed-cell sarcoma containing some large cells with several nuclei, which, however, were not the giant-cells of a myeloma. There was a fair amount of fibrous tissue, and in places chondrification was taking place. The growth was therefore a fibrifying and chondrifying mixed-cell sarcoma."

There was nothing of importance to note during the patient's convalescence. He attended one of the clinical evenings of the Society, when the tumour and microscopical specimens were exhibited. He has perfect movement of the ankle-joint, and no trace of any further growth or recurrence.

The main interest of this case depends on the fact that the tumour was a *primary* sarcoma of a synovial membrane. Such tumours are exceedingly rare.

Sarcoma may, of course, begin in the periosteum in the immediate neighbourhood of a joint, and subsequently involve

the synovial membrane, but nothing of this kind had taken place here. It was distinctly primary and absolutely limited to the serous membrane. The other structures of the joint and its surrounding were perfectly healthy.

The only other case of primary sarcoma of a synovial membrane of a joint that I know of is one published by Mr. Howard Marsh in the thirty-second volume of this Society's *Transactions*. The knee was the joint implicated, and a spindle-celled sarcoma was removed from the subsynovial tissue. It recurred in the scar after nearly three years. In all the disease was removed locally five times, and the thigh was eventually amputated. There were no secondary deposits.

Dr. Henry F. Bellamy (*Journal of Pathology*, vol. vii, page 465) has drawn attention to the myeloid tumours of tendon sheaths. In a table of twenty-three cases, two were connected with the tendons of the thumb, eighteen with those of the fingers, two with the toes, and one with peronei tendons; and quite recently, at the Pathological Society, Mr. Gask showed a specimen (probably an endothelioma) from the sheath of the tibialis anticus.

I myself have removed a myeloma from the tendon sheath of the finger of a young lady who had overstrained it by excessive violin practice. These tumours, growing from tendon sheaths, and, according to Bellamy, probably originating from the endothelium of the blood-vessels, are myelomata and innocent, and differ materially in microscopical structure and probably in clinical characteristics from the primary sarcomata of the synovial membranes of joints. Mr. Howard Marsh's case was of the spindle-celled variety, mine a mixed-cell sarcoma.

Looking to the repeated recurrences in different places of the synovial membrane that occurred in Mr. Marsh's case, one cannot look with confidence to the future of my own patient. The tumour, however, "popped" out so readily, and is so obviously entirely removed, that it is to be hoped he may escape recurrence.

XXV.—*A case of Sarcoma of the Synovial Membrane of the Knee.* By Charles Barrett Lockwood. *Read May* 9, 1902.

In October, 1894, a married woman, æt. 24 years, was under my care in St. Bartholomew's Hospital on account of disease of the right knee. Three years before, in 1891, the knee swelled and became painful without any known cause. The pain was slight but continuous. Starting pains were absent. She could not kneel, but was able to walk "fairly well." When she was admitted the joint was slightly flexed, but possessed almost its full range of movement, and the movement was painless except in extreme flexion or full extension. The swelling had obliterated the patellar fossæ, and was clearly confined to the limits of the fibrous capsule. It projected most upon the inner side, and partially obscured the outlines of the femur and tibia, but did not bulge into the popliteal space. The right knee was three quarters of an inch larger than the left, the circumference being measured at the upper end of the patella. The swelling felt rather warmer than other parts of the limb, and was slightly tender on pressure. The muscles were very little wasted, barely half an inch difference between the two calves. The body temperature was not raised nor the pulse accelerated.

On December 19 the patient was shown at consultations, and the general opinion was that the case was one of tubercle of the synovial membrane. But a doubt seems to have existed, for some one raised the question of syphilis of the synovial membrane.

Inasmuch as hardly any treatment had been attempted a knee splint and mercurial dressings were applied. After a while the patient went back to the country wearing a leather splint, and with instructions to return for observation and inspection.

Nothing was heard or seen of the patient for many months until she presented herself again in September, 1895. In the meantime the joint had gradually got worse, and became the seat of constant aching, now and then aggravated by startings. The right knee was slightly flexed and the tibia displaced backwards on the femur. The wasting of the

thigh amounted to three inches. The swelling at the right knee-joint had increased until the circumference of the joint was two and a quarter inches greater than that of its fellow. The swelling now completely obscured the outlines of the ends of the femur and tibia, and also the outlines of the patella. It did not project into the popliteal space. Its surface was slightly protuberant in places, rather hot to the touch, and exceedingly tender on pressure, especially around the patella. The skin over it was slightly reddened by hyperæmia, and some of the veins enlarged. The swelling now felt softer, and towards the inner side was thought to fluctuate. On admission the body temperature was 98° F. Three days later it rose to 99·6°, and next day to 102·1°, and then to 101·2°, after which it subsided to 98° again. I mention this fact because the rise of temperature helped to confirm the opinion that we still had to deal with an inflammatory affection of the synovial membrane of the knee. Also I think that it will be conceded that the local conditions were very strongly in favour of this diagnosis. If this be doubted let me recall the muscular wasting, the flexion of the knee, the displacement of the tibia backwards, the redness, pain and swelling, and the softening, too, at the inner side.

Acting on the assumption that the case was one of progressive tuberculous disease of the synovial membrane of the knee, I performed resection on September 25, 1895. The joint was laid bare by a semilunar incision which divided the ligamentum patellæ. The flap including the patella was turned up. This brought into view the mass of very soft material which was pronounced at the time to look "almost like sarcomatous growth." This entirely filled the capsule of the joint, and had destroyed the crucial ligaments, and part of the patella, and articular surfaces of the femur. Half of the patella and the whole of the condyles of the femur had to be removed before healthy bone was reached. The cartilage of the tibia was intact. The patella and femur were thought to be carious. But at this stage my suspicions were aroused by the complete absence of any caseous material or of any tuberculous abscesses. Some of the tissues were sent for pathological examination, and the late Dr. Kanthack reported that it consisted of "spindle-celled sarcoma." Mr. James Berry also examined another portion, and reported that it was "oval-celled sarcoma."

The result of this operation was most unfortunate. The operation lasted an hour and a half, and was attended by a

moderate amount of hæmorrhage, but followed by considerable shock and collapse. By night these had worn off, and her condition was, with the exception of pain, considered satisfactory. A morphia injection was given for the pain. Towards midnight the faintness and collapse recurred, and she suddenly died of syncope, eleven hours after the commencement of the operation.

A post-mortem examination was held, and Mr. Berry reported that no secondary deposits could be found, and that "all signs of disease had been completely removed from the knee." Also that the abdominal viscera were very bloodless.

Looking back upon the clinical history of this case of sarcoma, I believe that should another like it be seen I should again be deceived. The clinical notes, which were written before the nature of the disease was known, would describe almost any case of progressive tuberculous inflammation of the synovial membrane of the knee. I can only detect two clinical signs which might have excited suspicion. It may be remembered that when the patient was first seen in October, 1894, the swelling of the synovial membrane was already considerable. Now this was associated with two features which were hardly ever met with in the advanced stages of the tubercle of the synovial membrane: first, muscular wasting was almost absent; second, the joint was still capable of a wide range of painless movement. Perhaps, too, the history may have been misleading. Three years is a very long time for the growth of a soft sarcoma, but is quite in accord with the presence of tuberculous disease.

XXVI.—*A case illustrating some of the clinical features of "Horizontal" Dilatation of the Left Auricle.* By Wm. Ewart, M.D., and Sir Isambard Owen, M.D. *Read May* 9, 1902.

THE present case, which was exhibited before the Society on January 24th, 1902, is believed to be the first in which a clinical diagnosis was made of that extreme variety of dilatation of the left auricle which was introduced to our knowledge as a post-mortem finding in the remarkable case brought before the Society last year, and in which the enlargement is such as to cause the left auricle to approach the right infra-mammary surface beyond the boundary of the right auricle itself, and to pulsate in that region like an intra-pericardial aneurysm.

The name *horizontal dilatation* is perhaps that which will best identify this variety of major dilatation. The more common form is well known to pathologists. It expands the auricle symmetrically, but chiefly in the *vertical* diameter, tending to raise both main bronchi and perhaps to compress both pulmonary roots; whereas the horizontal dilatation takes place chiefly towards the right, and need not affect the left main bronchus or the root of the left lung.

A complete description of the clinical features of this affection is not yet possible. Nevertheless, as its earlier recognition and its earlier treatment are urgent indications, we have endeavoured to bring out, by comparing the clinical record of the original case (a record unbiassed by any knowledge of the possibility of this condition) and that of the present one, some of the broad lines which may in the future facilitate its identification. In so doing we make the reservation that, although we have reasons to regard the present case as an instance in point, this cannot be proved with absolute certainty.

Notes of the Case.

"C. W., æt. 32, male, omnibus driver for seventeen years, of moderate habits as regards alcohol, had been previously quite free from illness and ailments, except winter coughs and colds, and an attack of right-sided pneumonia in 1896. Rheumatism, chorea, scarlet fever, and syphilis were denied.

His present illness dates from a 'chest cold' caught one month prior to his admission into St. George's Hospital under the care of Dr. Ewart on November 20, 1901 (*Med. Reg.*, No. 1979, 1901)." In the interval he had kept at work (in spite of shortness of breath and palpitations during the last fourteen days) until November 19, when he was unable to get up. For fourteen days also he had begun to be propped higher in bed during the night; and there had been heavy sweats the last three nights.

On admission.—Face a little puffy, with bluish dusky lips and complexion, and external jugular veins filled, but not greatly distended or visibly pulsating. No ascites and hardly any anasarca. Pulse irregular, quick, with many small beats. Respiration quick but without much distress. Slight cough but no expectoration. The liver dulness extends from the sixth rib to one inch below the costal arch in the mammillary line, and there is marked hepatic pulsation.

Diminished movement and a weaker respiratory murmur are noted on the right side; there are coarse râles, chiefly at the bases, in both lungs. The cardiac impulse in the left præcordium is extensive. A visible and palpable impulse is also perceived below the level of the right nipple, and internal to the latter—chiefly in the fourth intercostal space. This area is the seat of the habitual pain described by the patient, and is, together with the epigastrium, the only tender part, the left præcordium not being tender or painful. The cardiac apex is felt to beat under the sixth rib, about one and a quarter inches beyond the left mid-clavicular line. By percussion it is found that *anteriorly* the cardiac dulness is much enlarged in its transverse diameter, extending from the right nipple to one inch beyond the left nipple, and somewhat also in its vertical diameter, the liver being depressed as well as slightly enlarged; whilst at the base there is no dulness suggestive of an aneurysm.

Posteriorly the dulness belonging to the heart is slightly enlarged, but that special to the left auricle is, as may be seen in the tracings, considerably increased (from a normal size of about 2½ inches by 2 inches to that of nearly 4 inches by 2½ inches), mainly in its transverse diameter and in the direction of the seat of abnormal pulsation, which, it is thought, may be identified by auscultation as belonging to the left auricle. Two murmurs may be heard at the apex—a slight and uncertain presystolic, and a distinct steady systolic murmur. The latter murmur is also audible over the

greater part of the cardiac area, but not far towards the left, whilst both murmurs are heard with almost undiminished clearness in the right inner mammary region.

The pulsation of the liver is truly expansile and very marked, but apt to disappear completely with rest. Dr. T. R. Whipham, Medical Registrar to St. George's Hospital, who examined the patient later in the day, made a definite note that there was then no hepatic pulsation.

The case was diagnosed after physical examination as one of major "horizontal" dilatation of the left auricle. For a while the patient was placed under simple treatment by rest, tonics, and stomachics, whilst operative measures had to be adopted for a rectal fistula and abscess, which healed before December 15.

On December 30, after several days of increasing flatulent distension of the abdomen, the patient woke with a feeling of something giving way in the third and fourth interspaces in the right parasternal line, and this sensation was immediately followed by hæmoptysis (about 12 oz.). Owing to his giddiness and faintness he was made by the nurse to lie flat in bed. This attitude was maintained during that day and the next, and resulted in much increased dyspnœa and cyanosis, for which he was bled to 16 oz. on December 31 with marked relief, the pulse dropping from 140 to 90. Later in the day, the raised attitude having been resumed, the cyanosis was much diminished. In addition to the signs previously mentioned he presented that day a distinct thrill near the right nipple and also at the cardiac apex. The veins in the neck were full and the liver again pulsating. Hypodermic injections of strychnine had been given, and some digitalis and ammonia were prescribed and taken for two days, after which no medicine was administered, the patient making satisfactory progress, and leaving his bed about January 7th.

He is now rather stronger and able to walk slowly on a level without any distress of breathing, and there is no orthopnœa or subjective cardiac symptom. But the pulse is still very uneven, and the small beats predominate. There is no facial congestion, but the lips are slightly dusky. The size of the cardiac dulness, of which tracings had been kept, is slightly diminished. The pulsation in the right fourth interspace is no longer perceptible, except under the stress of cardiac excitement from hurried walking or stooping.

Clinical Remarks.

It is warrantable to infer that in its extreme form this affection is decidedly uncommon, since the leading diagnostic feature—systolic pulsation with or without thrill in the fourth right intercostal space—is one so obvious and important as to preclude its having been habitually overlooked during life; whilst at autopsies the partial collapse induced at the base of the right lung might have called attention to the cardiac condition where it chanced to occur.

The *ætiology,* probably rheumatic in the first case, remains unascertained in the present one.

The *clinical history* is one of chronicity, with insidious onset and gradual development. Finally, right-sided pulsation must become obvious and may be recognised, or it may remain undetected; prostration may be induced either by the dilatation itself, or in association with some intercurrent cause of depression. During the long interval of development both patients were singularly free from severe symptoms, except some dyspnœa on exertion, and the present patient was not aware of shortness of breath until within a fortnight prior to his admission.

But for the remarkable toleration shown for this form of enlargement, the auricle could not grow to so great a size. Not only is its huge dilatation compatible with life, but neither in the shape of pain and distress of breathing nor in the shape of failure of compensation need it until quite late bring about a complete deadlock in the functions. Thus in the original case the patient had long intervals of comparative relief, and the present patient was able to continue his work and did not suffer much inconvenience until quite recently.

The *diagnosis,* which was previously impossible even at the latest period, may perhaps in the future be made by means of skiagraphy at a comparatively early stage, but not without the help of dorsal percussion, and in particular of percussion of the auricle. Hitherto we have not been aware of our opportunities in this direction. Dealing for the present only with the fully developed condition, we may call attention to the following objective and subjective indications.

The *leading objective signs* noted were—

(1) Systolic pulsation with or without thrill in the right fourth intercostal space.

(2) Synchronicity of this pulsation with the apex-beat.

(3) Equality in the loudness of the existing murmurs at the pulsating area and at the apex.

(4) Great increase in the transverse diameter of the præcordial dulness, which probably may extend even beyond the right mammillary line.

(5) Increase of the post-cordial dulness, with a characteristic asymmetrical enlargement (chiefly in the transverse diameter) of the post-auricular dulness. The general enlargement of the heart itself as well was clearly shown in the tracings of this case.

(6) Moreover it is noteworthy that the liver, as in this case, may be at times the seat of an inconstant pulsation. And again—

(7) The absence in this case, in spite of the cyanosis and dyspnœa, of any advanced general or serous dropsies is remarkable.

(8) The *dyspnœa* seems to present an important clinical peculiarity in its association with cyanosis and with quick breathing without any great respiratory distress. More striking still was the readiness of the present patient to lie almost flat, in marked contrast with the common type of orthopnœa seen in advanced cardiac cases.

(9) Lastly, an important confirmatory sign is the impairment of the respiratory movements and sounds on the right side of the chest.

The *subjective symptoms* were above all the pain and tenderness localised to the right of the sternum, in contrast with those special to ordinary cases of mitral stenosis, in which the pain is referred to the "ventricular," or left parasternal region. To the dyspnœa reference has already been made. In the present case the tachypnœa and the tachycardia rose to a considerable rate together with the cyanosis whilst the patient was recumbent; nevertheless the suggestion to sit up did not come from the patient himself, but from the physician, and this shows the tolerance displayed in this instance for the horizontal posture.

As regards prognosis and treatment, the inference is that the condition when fully developed is not likely to be cured. The observations in hand tend, however, to show that considerable alleviation may be effected by rest and by venesection rather than by medicinal treatment. It is probable that on those lines, had the condition been recognised earlier, the heart in both cases might for a longer time have been protected from the worst developments. The danger in these cases is their latency and their insidiousness.

XXVII.—*Horizontal Dilatation of the Left Auricle.*

By Sir ISAMBARD OWEN, M.D. *Read May* 9, 1902.

IN May of last year I showed a specimen of extreme horizontal dilatation of the left auricle of the heart, and communicated particulars of the case. It will be remembered

DILATATION OF LEFT AURICLE.

1259b. A Heart showing enormous dilatation of the Left Auricle, associated with mitral disease. The left auricle is larger than the rest of the heart taken together, and is equal in size to two closed fists. Its walls are much thinned, and contain calcareous plates on the left side. The left appendix auriculæ is scarcely, if at all, dilated. The outer surface of the auricle is injected, and shows at its upper part the remains of fibrous adhesions, the result of localised pericarditis. The mitral valve is not appreciably stenosed, but the valve segments are thickened and puckered, and the chordæ tendineæ are thickened. The left ventricle is dilated and hypertrophied. The right auricle is moderately dilated, as is its appendix. There is marked hypertrophy of the right ventricle.

From a married woman æt. 27 years, who had been repeatedly under treatment for mitral disease at the Greenwich Infirmary for a period of nearly five years before her death. A year before death the heart's apex was in the eighth intercostal space, 2 inches outside the nipple line. The action of the heart was irregular and tumultuous. There were loud presystolic and soft systolic bruits at the apex. Later, the apex-beat reached the mid-axillary line; the heart's dulness reached up to the third rib, and extended slightly to the right of the sternum; the double mitral bruit persisted. There was cyanosis, with much dyspnœa and œdema.

The report of the post-mortem examination states that, "on opening the thorax, the lungs were seen to be widely separately by the pericardium. On opening the latter, it appeared at first to be much too big for the heart, as though it had been greatly distended with fluid; but there was no abnormal fluid present. On raising the heart, the posterior surface of the pericardium was seen to be raised by what was apparently a large extravasation into the posterior mediastinum, but which afterwards proved to be the enormously distended left auricle, which had become doubled backwards, as it were, behind the rest of the heart. The auricle was entirely filled with recent clot. The mitral orifice easily admitted three fingers. The lungs were extremely emphysematous."

The mitral valves are a little thickened towards their free border, but there is no other appearance of disease.

The pericardium is universally adherent and separated from the muscle by a layer of fat, and three unabsorbed effusions of blood are found outside the heart and within the pericardial sac.

The symptoms were orthopnœa, cough, extensive œdema of the legs and sacral region, and ascites. The dropsy was stated to be of ten years' standing, the shortness of breath of twelve months', and the cough of three weeks' date. There had been a left hemiplegia five months previously.

The jugular veins were distended from above, and the pulse unequal and small.

The apical impulse was diffuse, in the sixth space and mid-axillary line; the area of dulness corresponded, and reached up to the third rib; there was none to the right of the sternum. There was no murmur.

The patient ultimately died of basic pneumonia.

There was no history of pericarditis, and the case was regarded during life as one of granular kidney.

In view of these two specimens mine cannot be looked upon as a mere pathological curiosity. It must, I think, be regarded as illustrating a definite though rare type of cardiac disease, for which my colleague, Dr. Ewart, has proposed the apposite name of "horizontal dilatation," so as to distinguish it from the spherical or "vertical" dilatation of the left auricle met with in mitral stenosis. As far as we know at present the condition is exceedingly rare, but it is not improbable that other though less extreme cases will before long be put on record if the attention of clinicians and pathologists is called to the possibility of its existence.

The time has hardly come to write the natural history of the condition. In a further joint communication Dr. Ewart and I have made some attempts towards it. I will at present content myself with summing up the points of coincidence between the three cases at present before us. For the sake of convenience I will denote my own case as Case 1, that from St. Bartholomew's as Case 2, and that from University College as Case 3.

In all these cases the direction of the dilatation is similar and remarkable. In place of expanding equally in all directions, the auricle is enlarged mainly to the right, passing behind the right or anterior auricle and appearing on the right border of the heart, but not rising much above its normal

upward limit. In my own case, as will be recollected, it had separated the right lung entirely from the diaphragm, and abutted against the vertical walls of the right side of the thorax in their entire circumference. The heart in all the cases was pushed over to the left.

In all three cases the pericardium is extensively adherent. In No. 2 it is slightly, and in No. 3 extensively calcified.

In no case is there any appearance of mitral stenosis, though the edges of the valve are somewhat thickened in all. In Case No. 1 the mitral orifice is dilated, but not so in the others.

In all the right auricle is also dilated, though to a less extent than the left. In none is there any disease of the tricuspid orifice.

The ventricles are hypertrophied in Cases 1 and 2. In No. 2 the left, and in No. 1 both ventricles are dilated. In No. 3 they are neither hypertrophied nor dilated.

In all three the leading symptoms of the fatal attack were severe dyspnœa and a serious degree of œdema. In Nos. 1 and 3 it is said to have affected the entire lower extremities, in No. 2 its area is not recorded. In No. 3 only was there ascites, and in this case only was engorgement of the jugular veins recorded.

In Cases 1 and 2 the apex-beat is recorded as having been in the eighth interspace—a most unusual situation in any other form of heart disease. In No. 3 it was only in the sixth.

In Case No. 1 it was as far back as the posterior axillary line, in the others only in the mid-axillary.

In all three the cardiac dulness involved the third left space, but in No. 1 alone there was dulness, and that only slight in the second.

In Case No. 3 the heart-sounds were natural, in the others both systolic and presystolic murmurs were heard at the apex; the second sound was sharply heard at the apex in Case 1, its absence is not recorded in Case 2.

In Case 1 alone were the obvious signs of a pulsating mass of fluid in the right chest present. In Case 3 there was no extension of dulness to the right of the sternum.

The evolution of the condition had in every case been the work of many years. In No. 1 only was a history of rheumatic fever recorded. In No. 3 the patient had had no rheumatism.

Two of the three cases were in women. The ages of the patients vary from twenty-seven to sixty.

XXVIII.—*A Note on Palpable Systolic Intercostal Pulsation over the Right Auricle, and on its Occurrence in a Fatal Case of Mitral Stenosis, with Dilatation and Thrombosis of the Left Auricle.* By Wm. Ewart, M.D. *Read May* 9, 1902.

THE pathological and the clinical history attached to the heart now exhibited might alone justify this brief communication. The left auricle was markedly dilated above a button-hole stenosis of the mitral valve, and was completely filled with a spongy thrombus which had partly broken down into a grumous fluid; and the case presented clinical features of interest in the intense unrest and depression of the patient, in the cyanosis without severe orthopnœa, and in the tachypnœa without much acceleration of the pulse. But the paper has a more limited scope. Its special object is to elicit further information concerning the significance of systolic pulsation felt over the right auricle as a diagnostic physical sign, and concerning the frequency of its occurrence and its various modes of causation. In this instance the pulsation was wrongly diagnosed during life as the direct pulsation of an enlarged left auricle, evidence of left auricular enlargement having been obtained by percussion in the back. But it was found after death that the seat of the pulsation corresponded to the surface of the right auricle, and that the left auricular dilatation was not of the extreme degree nor of the horizontal type which had been diagnosed.

Notes of the case.—Teresa D., general servant, æt. 24, single, was admitted into St. George's Hospital (Med. Reg. No. 2189, 1901) on December 21, 1901, after an illness of six weeks. Previously strong, she had become short of breath since an attack of rheumatic fever two years ago, but had kept at work. Though fairly nourished, she was pale, cyanosed, and prostrate, and breathed rapidly, but without much respiratory distress. The tachypnœa and cyanosis were not apparently due to bronchial obstruction, for the bronchitis was slight. Further peculiarities were the disturbed nights and the somnolence all day, the patient's peevishness,

the tendency to sighing breathing when any one came near, and her perpetually distressed and unhappy aspect and utterances. An occasional contracture in one foot also suggested to several observers the view that she was inclined to hysteria. But the most important feature was the disinclination for food, owing to which, on one occasion, my house physician thought it desirable to pass the nasal tube. The patient did not habitually assume the sitting posture, more generally she was reclining with not more than two pillows under her head. The pulse was quickened (varying between 100 and 120) and uneven. It subsequently became regular under treatment, and during the last three weeks it hardly rose above 88. The respirations were accelerated throughout, and particularly during the last week, when they averaged 44 per minute. There was little œdema of the legs when she was admitted, and this entirely disappeared, but it finally returned. The urine was free from albumen. The cardiac area showed an extensive visible impulse. The apex-beat could be felt some distance to the left of the normal position, whilst the right border of cardiac dulness extended beyond the normal limit, and the liver was slightly depressed, as an additional evidence of the marked cardiac enlargement. Inequality of the respiratory movements was easily noticeable, the right side of the chest moving less than the left. No thrill was noted, but a presystolic murmur was heard, in addition to a long systolic murmur, and the case was regarded as one of mitral stenosis. The external jugulars were visible, but not apparently greatly distended. My own examinations had been cursory, and had not succeeded at first in clearing up to my satisfaction the unusual points in the case.

On January 22, after a short absence, finding the patient weaker, whilst a tendency prevailed to regard her distress as in some measure assumed, I made a more searching physical examination, with the result now to be described. Anteriorly the liver was depressed, somewhat tender, but not pulsating. The cardiac dulness presented a much increased transverse measurement. A systolic pulsation and great tenderness were noted to the right of the sternum, coinciding precisely with the border of the dulness, the outline of which was not as regular as that of a normal right auricle. Great tenderness was also present at the epigastrium, where the heart could be felt beating. Over the left part of the precordium there was no tenderness. Posteriorly the cardiac dulness was increased in size; but much more increased

relatively was the dulness of the left auricle itself, the extension being chiefly in the transverse diameter and to the right of the middle line. Here, again, the right boundary of dulness did not present the clean crescentic outline of a normal right auricle, and I was led to think that a part of that dulness, which appeared to be right auricular dulness, probably belonged to a dilated left auricle stretching forwards and towards the right from the posterior surface.

On careful palpation of the præcordium a systolic intercostal impulse was felt between the right nipple line and the sternum, and in this situation the heart-sounds and murmurs were heard as loudly as at the apex; whilst posteriorly, on direct auscultation over the area of dulness described, it was possible to perceive not only the distant heart-sounds, but a distinct systolic cardiac thud. The similarity of these physical signs with those observed in another patient, who was simultaneously under my treatment, led me to conclude that they had the same derivation in both cases, and were due to considerable horizontal enlargement of the left auricle.

The symptoms showed no improvement under treatment, and the patient never rallied from her depression. During the last two weeks she repeatedly said that she was dying. On January 25 the hands began to swell. On the twenty-seventh she was bled to 14 oz., to relieve her increasing dyspnœa, but without success. On January 30 both feet were incised, and about 6 oz. of serum escaped from the incisions each day. In spite of this she lost strength rapidly, and died of exhaustion on February 2.

At the *post-mortem* examination fluid was found in both pleuræ, and a pleuritic false membrane in the right pleura. The lungs were congested, and contained infarcts. The right auricle was dilated, and the right ventricle moderately dilated. The tricuspid valve was stenosed, its diameter measuring two-thirds of an inch. The condition of the left side of the heart has already been described.

Remarks.—Visible or palpable pulsation of the right auricle does not, in the writer's experience, belong to the normal cardiac function; if capable of being felt, the beat should presumably be presystolic, not strictly systolic in time. In the ordinary cases of dilatation of the right side of the heart, he has not hitherto noticed any pulsation, whether presystolic or systolic, to the right of the sternum. When the heart happens to be displaced into the right chest a pulsation is felt in the right intercostal spaces; but the abnormal pulsa-

tion is then systolic, and for anatomical reasons it must be regarded as a direct ventricular pulsation.

If palpable systolic pulsation over the right auricle is as uncommon as the writer believes it to be, and if systolic pulsation of the right auricle, howsoever greatly this auricle may be distended, is never observed as a mere result of dilatation, its occurrence would suggest in every case the influence of some additional factor besides simple dilatation of the right auricle, and any definite observations on this point by others would be of much value. Systolic pulsation within the auricular district of the præcordium has now been shown by Sir Isambard Owen to be sometimes directly produced by the left auricle when this is immensely enlarged. The present case demonstrates another fact, viz. that a systolic pulsation is capable of being transmitted to the infra-mammary intercostal spaces through the right auricle when the left auricle is kept distended in such a way as to render it unable to perform its systolic contraction. To these two modes of causation for the systolic auricular pulsation in question others may possibly be added in the future as the result of further inquiry.

A case which seems to be highly suggestive of the former of these two conditions was contributed by Dr. J. Porter Parkinson to the last volume of the Society's *Transactions* (p. 221), with a diagram illustrating the cardiac dilatation, the position of the systolic thrill in the right infra-mammary region, and the enlarged pulsating liver. That observation may eventually acquire yet greater value, and may throw further light upon the practical point which forms the subject of this communication, for it is stated that "the slight anasarca disappeared rapidly. Since November the heart has somewhat lessened in size, but otherwise there is little change. The patient has never had any marked cardiac symptoms."

Parkinson inquires whether the cardiac disease may not have been of intra-uterine origin, there being so little distress. His conclusion is that there is great enlargement of the heart, especially of the right auricle, and double disease of the mitral and of the tricuspid valves. The comparative absence of symptoms is remarkable, there being hardly any cyanosis and dyspnœa, except with exertion. The liver pulsation is well marked, but there is scarcely any epigastric pulsation or engorgement of the jugulars.

The tracing of the precordial dulness in Parkinson's case

closely resembles the tracings taken by myself from a patient whose case Sir Isambard Owen has joined me in reporting. It seems not unlikely that both cases may be of the same kind, and that in Dr. Parkinson's case the systolic pulsation and thrill may belong not to the right auricle, but to a greatly distended left auricle approaching the anterior chest wall. There are these further resemblances, that in our patient also the chest is unusually deep, that there had been a remarkable absence of cardiac symptoms, and that the patient's condition rapidly improved under rest and treatment. At the present date (middle of March, 1902) the pulsation has ceased to be felt, except when the patient is caused to move about rapidly, and the general improvement is maintained.

XXIX.—*Inheritance of Recurrent Attacks of Jaundice and of Abdominal Crises, with Hepato-splenomegaly.*

By Sir THOMAS BARLOW, Bart., M.D., and H. BATTY SHAW, M.D. *Read May* 23, 1902.

THE observations giving rise to this paper have been made upon two patients, Albert W., æt. 19½, and his mother, Mrs. W., æt. 47, and they are recorded as illustrating the inheritance from mother to son of enlargement of liver and spleen, with recurrent attacks of jaundice.

Albert W. first came under our notice in October, 1899, being then sixteen years old. The mother stated that when the child was born he was pale and waxy, but not jaundiced. She states that the doctor said that the boy's liver and spleen were both enlarged, and that the heart was abnormal. He was very delicate during the first year of life, and a little brandy was given three or four times a day during the first year. He was observed to be jaundiced for the first time when he was five years of age, but since that time there have been attacks of yellowness varying in intensity from day to day; in these attacks there was much pain on the right or left side of the upper part of the abdomen. There was no shivering and no vomiting; no change of colour was seen in the motions, but the urine was observed to be darker than usual, and a thick pinkish deposit formed. The attacks lasted three or four days. Six years ago the patient began to be troubled with ulcers on the outer malleolus of the right foot and the inner malleolus of the left; they slowly healed with rest, and recurred when he got about again. The only other illness from which the boy has suffered was a doubtful slight attack of measles.

On admission, the patient was found to be a slightly built youth, and distinctly under-sized for his age; he was anæmic and yellow, and the yellowness became deeper for a few days after admission, but when most marked it was still only of a lemon tint. The liver was only moderately enlarged, never extending more than three fingers' breadth below the costal margin; the edge was difficult to feel, and the surface, so far as it could be felt, gave no suggestion of induration or undue

convexity; the upper limit in the right nipple line was in the sixth space. The spleen was enlarged, and caused some bulging of the lower part of the left chest; it extended to two inches below the level of the umbilicus, but did not cross the middle line; there was a well-marked notch in the anterior border, and the whole organ moved freely with respiration. There was no ascites, and the abdomen was otherwise normal. The apex-beat was situated in the fifth space, half an inch external to the nipple line; there was no thrill, the impulse was not heaving in character; there was a hæmic murmur heard over the pulmonary cartilage, and a soft systolic murmur at the apex conducted for a very slight distance outwards, which was considered to be due to slight dilatation of the left ventricle. There was slight pulsation in the veins at the root of the neck. There was no clubbing of the fingers or toes. The lungs were normal. The tongue was slightly coated, flabby, and pale; the gums were very pale. The stools, repeatedly examined, were always brown in colour. At no period was itching present. There were indolent ulcers situated one over the right external malleolus, and one over the left internal malleolus, and there was surrounding pigmentation, and a few small dilated veins in the neighbourhood. There was no evidence of syphilis, congenital or acquired. The urine was high-coloured, containing a large deposit of pink urates, specific gravity 1025; there was no albumen or sugar; no bile was found, but urobilin was abundant. The blood was examined, and it was found that the amount of hæmoglobin was 60 per cent. of the normal, and the red corpuscles were reduced to 3,475,000 per cubic millimètre. During the twenty-one days the boy was in the hospital his highest temperature was 99·6°; it was generally about 99°, and occasionally subnormal. The jaundice, after deepening for a day or two, subsided, but the boy was still slightly yellow when he left the hospital, and the character of the urine remained the same; the heart condition improved with the rest in bed; the ulcers healed; the spleen remained unchanged, but the liver diminished in size.

During the next six months the boy had at least one abdominal attack, in which he became much more yellow, and had dull, heavy pain over his liver; he was ailing for a fortnight, and for three days of this fortnight he was in bed. It was at the end of this attack, viz. on May 3, 1901, that he was readmitted.

There was practically no change in the size of the spleen,

and the lower edge of the liver did not extend below two fingers' breadth from the costal margin. The cardiac condition was practically unchanged; there was enlargement of some of the glands of the neck, but this subsequently subsided. The temperature was normal or subnormal during the month that he was under observation. A detailed examination of the blood and urine was made, and the results are given in the following tables (Tables I and II):

TABLE I.—*Examination of Urine.*

Date.	Quantity in c.c.	Colour.	Reaction.	Specific gravity	Total urea.	Urobilin.	Remarks.
1901.	c.c.				Grms.		
May8	780	Dark	Acid	1024	17·55	—	First treated with Liquor Arsenicalis miij ter die.
9	1020	„	„	1024	21·42	Urobilin on extraction.	
10	1110	Very dark	„	1020	20·52	„	Very much urobilin.
11	960	Dark	„	1020	12·48	„	
12	1095	„	„	1020	17·52	„	
13	975	„	„	1021	18·52	„	
14	1125	Very dark	„	—	22·50	„	
15	960	„	„	1022	20·16	„	Arsenic omitted.
16	1065	„	„	1022	14·91	„	
17	960	Dark	„	—	17·28	„	
21	990	—	„	1021	—	„	
22	735	—	„	—	12·49	„	
23	900	Dark	„	—	18·00	„	
24	840	„	„	—	—	—	Inunction with mercury, 1 drachm daily.
25	697	„	„	—	13·94	Urobilin on extraction.	
26	435	„	„	—	9·13	„	
27	690	Very dark	„	—	12·42	„	Urobilin without extraction
28	667	Normal	„	—	14·34	„	
29	735	Dark	„	1022	12·49	„	
30	—	„	„	1022	—	„	

Bile in very minute traces was found on one or two occasions.

TABLE II.—*Blood Examination.*

Date.	Total red corpuscles per c.mm.	Poikilocytosis.	Total white corpuscles per c.mm.	Analysis of white corpuscles in percentage.					Nucleated red corpuscles.	Basophile leucocytes.	Time of clotting.	Hæmoglobin percentage.	Remarks.
				Polymorphonuclear.	Coarse eosinophiles.	Large lymphocytes.	Small lymphocytes.	Transitional cells.					
									For every 100 white		Min.		
1901. May 8	2,587,500	Yes	14,062	71·5	5	7	16	·5	2	0	2	50	Liq. Arsenic. begun May 4th, 1901.
9	1,806,250	„	12,543	61	3	10	20	·1	3	Yes	2	46	
10	2,787,500	„	15,150	81	2	2	15	·0	2	„	2	42	
11	2,381,250	„	14,000	70	2	10	16	·2	0	0	3	36	
13	2,106,250	„	10,970	48	4	9	39	·0	1	0	1½	34	Blood exam. at 11 a.m. instead of 9 a.m.
14	2,037,500	„	14,050	67	1	10	22	·0	0	0	1½	34	
15	1,881,250	„	4,691?	66	5	12	16	·1	4	0	1½	34	
16	2,093,750	„	9,036	68	4	11	16	·1	3	Yes	3¼	34	Arsenic omitted.
17	1,806,250	„	9,389	70	1	7	17	·5	1	„	2	32	
24	—	„	14,000	68	2	1	27	·2	6	?	1	40	Inunction of Hg.
25	2,512,500	„	6,250	66·6	2·6	2·6	25	2·6	0	?	1½	38	
27	3,200,000	„	7,824	67	1	5	25	2	1	Yes	1½	38	
28	2,937,500	„	4,692	63	1	4	28	4	1	„	1½	48	
29	5,362,500?	„	6,250	67	1	3	28	1	2	?	1½	50	
30	3,300,000	„	6,250	56	3	7	29	5	1	Yes	2	48	
31	3,031,250	„	6,250	50	2	11	35	2	2	„	—	46	
Avrg.	2,603,753	—	9,793	65	2·4	6·8	23·3	1·8	1·8	—	1·85	41	

The above blood examinations were, with one or two exceptions, taken at 9 a.m.

The leucocytes were stained by Jenner's method, or, after being fixed by heat at a temperature of 150° C. for ½ hour, stained with the tricolour mixture of Ehrlich-Biondi-Heidenhain. Dahlia was used for the detection of basophile cells.

Dietary throughout stay in hospital:—Fish, chicken, eggs, bread, two pints of milk, one pint of beef-tea, tea, pudding.

Though the patient was slightly yellow, urobilin could not be demonstrated in the urine, except on two occasions, unless extracted by means of ammonium sulphate; only once or twice could a trace of bile be demonstrated in the urine. Very frequently, though not constantly, there was a copious deposit of highly coloured urates, those samples of urine which were free from deposit being usually the ones passed in the afternoons; the specific gravity of the urine was normal; the urine was always acid, and free from albumen, blood, and sugar. The total output of urea varied very much, being considerably more during the first twelve days of the patient's stay in the hospital, and whilst under treatment with Liquor Arsenicalis ♏iij given three times a day; during the latter part of the treatment, and when a drachm of mercurial ointment was employed daily, the output of urea fell. The patient throughout his stay in the hospital was on full diet (*vide* Table I).

With regard to the blood examination, the condition generally found was that met with in cases of secondary anæmia: at first the number of red blood-corpuscles per cubic millimètre was very much below the normal, whilst leucocytosis was very marked, and involved all forms of white corpuscles; later on, the number of red corpuscles increased somewhat, and the leucocytes decreased. Poikilocytosis was always present, and nucleated red corpuscles and basophile cells were frequently met with; the time of clotting varied from one and a half to three and a quarter minutes.

The patient was readmitted to University College Hospital in November, 1901, for the sake of giving the ulcers of the leg a chance to heal once more. The physical signs were practically identical with those already described.

Whilst Albert W. was a patient in the hospital in 1899 Mrs. W. used to visit him, and Mr. Arthur Ricketts, the house physician in charge, noticed that she had a yellowish complexion very similar to that of her son, and, as a result of this discovery, the following observations were made:

Mrs. W. had an attack of severe jaundice when quite a child, about four or five years old, and since marriage has had repeated attacks at varying intervals. These attacks have been accompanied by considerable pain in the abdomen, especially the upper part, and sometimes more on the right side, sometimes more on the left; vomiting also occurred in the attacks. Whilst pregnant with the child whose history has been given, she was seized with one of these attacks; no more attacks occurred for several years; about three years

ago she had another attack, and this has been followed by a number of others.

Through the courtesy of Dr. Bernard Reynolds, observations were made by one of us towards the end of an attack which occurred in September of last year. The patient had had for some days severe abdominal pain, occurring in paroxysms, accompanied by sickness, and very suggestive of biliary colic; there had been no fever, nor were any gall-stones discovered in the motions, which were pale but still slightly coloured. The patient was found to be a rather thin subject, and the skin was slightly icteric, but had been very much more highly coloured at the beginning of the attack; there were no abnormal signs in the lungs or heart; the liver was found to be considerably enlarged, both to percussion and palpation; the spleen, too, was obviously enlarged, reaching a few inches below the costal margin; there was slight varicosity of the veins of the legs, and on the right leg there was a small ulcer, which had caused considerable annoyance by constantly discharging during the last twenty-seven years; the ulcer followed an injury, and dates from before her marriage; there was no clubbing of the fingers, no ascites or œdema, nor was the pulse-rate reduced. The examination of the urine was negative as regards the presence of albumen, blood, bile, or excess of urobilin; during the earlier days of the attack the urine was much darker, but no observations were made on the presence or absence of bile or excess of urobilin. Blood examination showed that hæmoglobin was present to the amount of 50 per cent.; the red corpuscles numbered 3,993,750 per cubic millimètre, the white corpuscles 18,750 per cubic millimètre. Analysis of the white corpuscles showed small lymphocytes, 10 per cent.; large lymphocytes, 4 per cent.; transitional cells, 5 per cent.; polymorphonuclear cells, 75 per cent.; and coarse eosinophile cells, 6 per cent. There was well-marked evidence of poikilocytosis, and a few basophile cells were met with, but no nucleated red corpuscles. Clotting occurred in three minutes.

The patient is a very temperate woman, and has always resided in England; in her youth she had had smallpox, measles, and scarlet fever; there is no evidence whatever of luetic infection; the catamenia ceased at the age of thirty-four years. The rest of Mrs. Ws.' children, two girls and a boy, are quite healthy. The boy had an attack of jaundice once in late infancy; on examination, no enlargement of the

liver and spleen can be made out. The boy is quite healthy-looking. Another child, a girl, died in infancy, of convulsions.

Remarks.—These cases are brought forward for consideration for several reasons.

1. The occurrence of this disease in mother and son suggests the transmission from parent to offspring of some morbid process capable of producing in both almost identical symptoms. The only reference we can find as to the inheritance of such a condition is contained in a paper contributed to this Society by Dr. Claude Wilson (1). He describes a family in which six individuals, forming members of three generations, were subject to symptoms very like those described in this communication, and one of us has had the opportunity of seeing some of these cases, and watching their progress.

In the *Glasgow Hospital Reports* for 1899 Dr. James Finlayson (2) calls attention to the occurrence in members of the same family of conditions very similar to those described above, and considered by him to be examples of Hanot's cirrhosis of the liver occurring in children. He mentions four children in one family who were jaundiced: one, a youth æt. 18, had great enlargement of the liver, with jaundice, marked itching of the skin, and epistaxis; the urine was dark, and gave a slight bile reaction with iodine, very indistinctly with nitric acid or not at all; no distinct enlargement of the spleen was present, a very definite tumour in the left hypochondrium was regarded as the left lobe of the liver; he had also had previous frequent attacks of feebleness and ill-health, and was much undersized for his age. A sister, two years older, suffered from feeble health for seven years; she was also jaundiced, and had epistaxis and pruritus; she was ill-developed, and showed a great enlargement of the liver and of the spleen—the splenic tumour being more striking than that of the liver. A younger brother, æt. 15, was found to have slight jaundice and occasional itching and epistaxis; the liver reached three inches below the costal margin, the spleen three and a half inches. Another younger brother appeared to be a little yellow, but there was no enlargement of the liver or spleen. Syphilis and alcoholism could be excluded from the family history. Dr. Finlayson gives references to other cases where several collateral members of a family were afflicted with similar symptoms.

2. In reference to the two cases here described, the malady appears to have lasted certainly in the son for fourteen years, and may have been congenital, probably in the mother for a much longer period, though with some long intervals between the attacks. The same chronicity is noticeable in Dr. Wilson's cases (*loc. cit.*).

3. The occurrence of ulcers in the lower extremities which are so indolent in character points possibly to some abnormal blood condition. Dr. Finlayson (*loc. cit.*) describes in one of his cases a tendency to the formation of little abscesses, and in the case referred to by Dr. Roger-Smith (3) of a medical student, multiple chronic ulcers of the whole body constituted a marked feature.

4. The jaundice is apparently always slightly present, and undergoes great exacerbation periodically, and is then associated with severe pain about the liver or spleen, or both.

5. The occurrence of this malady in two patients related to one another raises the question as to the relationship of the various cases now grouped under the heading of Hanot's hypertrophic or biliary cirrhosis.

Mrs. W. might possibly be considered an example of this disease, but we are not convinced that in the case of her son any actual cirrhosis has, as yet, supervened. It is important to remember the group of cases occurring especially in children, known under the designation first employed by Banti (8 and 9) of splenic anæmia. In such cases the liver is often enlarged as well as the spleen, and both enlargements may ultimately subside. Also in the single case of Dr. Wilson's group, in which a *post-mortem* was obtained (10), the liver was found to be smooth, pale, and enlarged, but there was no increase of fibrous tissue; fat globules and pigment were found in the liver cells, which were not otherwise much altered. In the absence of *post-mortem* evidence, we think it best to limit our nomenclature to the clinical description we have adopted in this paper as "Inheritance of Recurrent Attacks of Jaundice and of Abdominal Crises, with Hepato-splenomegaly."

6. Further remark may be made in respect of these cases, and that is, to call attention to the extremely guarded prognostic view to be held in such cases. In the case of Mrs. W., above described, she has not only grown to maturity, but has had a family of five children, and all the four living members except one are quite healthy; and, except for the occasional periodic attacks of jaundice and abdominal crises, she is able

to enjoy life. It may be also mentioned that two of the boys described by Dr. Wilson have practically recovered.

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2. Dr. JAMES FINLAYSON, *Glasgow Hospital Reports,* 1899, p. 39.
3. Dr. HUGH R. ROGER-SMITH, *Clinical Society's Transactions,* vol. xxxi.
4. HANOT, *Etude sur une forme de cirrhose hypertrophique du foie,* Paris, 1876.
5. GILBERT FOURNIER et SURMOUNT, quoted in Brouardel and Gilbert's *Traité de Médecine, t.* v, 1898, pp. 351 and 522.
6. GILBERT et CASTAIGNE, *Société de Biologie,* 11me Série, i, 1899, p. 403.
7. Dr. FREDERICK TAYLOR, *Guy's Hospital Reports,* vol. lii.
8. BANTI, quoted by Brühl (q. v.).
9. BRÜHL, *Archives générales de Médecine,* 1891, No. 27, p. 673; No. 28, p. 160.
10. Dr. CLAUDE WILSON and DOUGLAS STANLEY, M.B., *Clinical Society's Transactions,* vol. xxvi, p. 163.

XXX.—*On the Subsequent History of Three Cases of Colitis on whom Right Lumbar Colotomy was performed several years ago.* By W. Hale White, M.D., *and* C. H. Golding-Bird. *Read May* 23, 1902.

IT may be within the memory of some members of this Society that on May 12, 1899 (*Clin. Soc. Trans.*, vol. xxxii), we read a paper here on three additional cases of right colotomy for chronic colitis. This evening we propose to give the subsequent history of each of these cases, the early details of which will be found in our previous paper.

Case 1.—Miss F., æt. 36, had suffered very severely from membranous colitis for twenty years. On May 13, 1896, the first stage of right lumbar colotomy was performed, the bowel being opened six days later. In May, 1897, a year from the operation, we closed the artificial anus without difficulty. During this year, from May, 1896, to May, 1897, her general health had considerably improved, and she had gained flesh. Her doctor, Dr. A. E. Taylor, of Lancaster Gate, wrote on August 4, 1896: "She has done very well indeed; not had a bad symptom; very few casts; she is now up and about, and is getting stout." After May, 1897, the date of closure, our first report is November, 1898, when she came to see us. She then looked perfectly well, was living an ordinary home life, was active in her habits, and taking moderate outdoor exercise, but had to take aperients to get the bowels open. From November, 1898, we heard nothing of Miss F. till we wrote to her in February, 1902. She was then in bed with rheumatic fever, so her mother wrote, saying as follows:—"She has to admit that there is very little change in her condition (compared with that before operation), and she could not candidly say there is a perfect cure." It is clear from this report that she has relapsed from her improved condition in November, 1898. Her mother continues her letter, saying, "The general health is very bad; occasionally swelling attacks (tympanites); great wasting of the body; the food passes through undigested, accompanied by casts and blood occasionally. . . . There is considerable constipation."

Dr. A. E. Taylor corroborates this written account, and he adds that she has recently had two attacks of subacute rheumatism. We wrote to the patient's mother to ask when the relapse began, and she replied that it was in December, 1898.

Case 2.—Laura B., æt. 31, a barmaid, had suffered from symptoms of severe colitis for eighteen months, passing much membrane. On March 3, 1898, the first stage of right lumbar colotomy was performed, the bowel being opened on March 8. The artificial anus was closed without difficulty on September 29, 1900, two and a half years after it was made. During this two and a half years in which the wound remained open, the patient remained in perfect health, resuming her business as a barmaid six months after the artificial anus was made, and subsequently continuing her occupation without interruption. She suffered no inconvenience from the protective apparatus she wore over the opening, and indeed twice refused our offer to close the artificial anus, as she had got quite used to it, and was afraid of running the risk of return of the membranous colitis. During the eighteen months that have now elapsed since the closure of the wound she has remained in perfect health, following her usual occupation. There has been no trace of membranous colitis, and to assure ourselves, last November we took her into the hospital for a short time and examined all the motions; they were perfectly natural. She remains well at the present time.

Case 3.—Edward K., æt. 35, had suffered from symptoms of chronic colitis for seven years. Colotomy was performed December, 1898. Five days later the bowel was opened, and seven weeks later he left the hospital to return to work much improved in his general health, and having lost all his symptoms of colitis. During the summer of 1899 he worked hard as an artisan in Woolwich Arsenal, and as he continued perfectly well was anxious for an early closure of the wound. Two attempts were therefore made during this year (1899), but neither was successful, though the opening was much reduced in size. Since the second of these attempts, very nearly all the fæces have passed naturally through the colon and *per rectum* in a perfectly healthy way, only a little occasionally coming through the colotomy wound. A third and last attempt was made in 1901, but again with only partial success. But on February 25, 1902, he presented himself in

good health, and reported that the small sinus resulting from the last operation had closed by itself. It will be noticed that he has remained well for two and a half years after the passage of fæces along the colon has been re-established.

We think these records show that in severe cases of chronic colitis which have not yielded to other treatment, right-sided colotomy is beneficial. Indeed, in each of these three cases it was curative, for Miss F. did not relapse until eighteen months after her wound was closed, and the other two patients, although their colotomy wounds are closed, remain perfectly well. The difficult question that has to be determined is how long shall the artificial anus be kept open, and whether in certain cases it should ever be closed at all. It is to be noticed that in Miss F.'s case the artificial anus was closed at the end of twelve months, during which period she had remained free from her symptoms, as indeed she did for several months after the closure, but eventually she relapsed. With our present experience, the extreme duration (20 years) and great severity of the disease in Miss F., who had become a thorough chronic invalid, would lead us to suggest, if we saw another patient like her, that the artificial anus should be maintained considerably more than a year; and though in our second case the artificial anus was kept open two and a half years at the patient's own request, it is likely that by this request she did herself a great service, for had we closed the wound earlier she might have relapsed; still, the duration of her disease was short. These two women are instances of *membranous* colitis. The third case, a man, was one of chronic dysentery. He so immediately improved after colotomy that we made the first attempt to close his opening in a few months, and we felt warranted in the attempt because, owing to the mechanical difficulties of plugging the wound in his cæcum, there were frequent escapes of fæces into the colon, although the passage of these was not sufficient to provoke any return of his symptoms. The difficulty in restraining the escape of the intestinal contents along the colon on the one hand and externally on the other, because of its fluidity, compels us to form an adverse opinion of cæcotomy in these cases as compared with a right lumbar colotomy; and for the same reason also a cæcotomy wound is more difficult to close.

To avoid the nuisance of an artificial anus, a small valvular opening in the colon has been suggested and employed (*Transactions of New York Surgical Society*, January 23,

1901, communicated by Dr. Percy Bolton). Through it antiseptic irrigation from it to the anus has been practised, but our opinion is that in membranous colitis irrigation does more harm than good; the complete rest given to the colon by the artificial anus is all that is required. Our reasons for this view are given in our former communication.

It has been suggested to us that the implantation of the ileum into the sigmoid would meet all requirements without an artificial anus, and ensure permanency of cure. Three objections at once present themselves to this plan. In the first place the irritating fluid contents of the ileum, passing at once into the rectum, would be likely to produce a chronic diarrhœa; secondly, some of the contents of the small intestine would be sure to regurgitate from the sigmoid flexure into the colon, and so keep up the colitis; and, thirdly, the implantation would, in many instances, not be above the whole of the disease.

CLINICAL CASES

EXHIBITED.

*** Published in accordance with the regulation relating to the exhibition of patients at the meetings of the Society, viz. that each case shall be accompanied by a brief written description of the points it illustrates; such description to be published or not in the *Transactions* as the Council may direct.

I.—*Right-sided Hemiparesis with Atrophy of Left Optic Disc.* By RAYNER D. BATTEN, M.D., and LEONARD GUTHRIE, M.D. *Exhibited October* 25, 1901.

BOY, æt. 12, fractured left femur in October, 1900. Whilst laid up had a doubtful attack of acute rheumatism. In February, 1901, noticed weakness (gradual) of right arm and legs, difficulty in writing and walking. When seen on April 30, 1901, weakness, ataxy, and intention tremors noted in right arm. Right leg wasted, knee-jerk exaggerated, ankle-clonus marked, and typical Babinski's plantar reflex present. Sensation normal. Gait hemiplegic in type. No facial paralysis. Faint presystolic bruit present. Veins of left optic disc engorged, but no swelling of disc.

May 21.—Left optic disc hazy, veins engorged. Vision not tested.

June 28 (Dr. Batten's note).—"Left optic disc pale and excavated. Cribriform plate exposed at bottom of excavation. Disc not filled in nor opaque. No blurring of vessels nor of margins of optic disc. Well-marked central scotoma for

white (no colour perception). Retina in macular area and up to optic disc showed signs of disturbance and a number of fine white dots. Veins somewhat large, dark, and tense." The right optic disc was normal. No hemianopsia.

October 1.—Paresis much improved, but still some intention tremors of arm, and increased reflexes in leg with extensor response in right great toe. Vision: left eye very slightly impaired; discs still pale, and veins rather large.

Remarks.—Nature of case is obscure. Patient may have had thrombosis in neighbourhood of left motor tract with retro-bulbar neuritis of left optic nerve.

May be compared with case shown at Clinical Society's meeting, October 28, 1898, vol. xxxii, page 228, in which complete left hemiplegia and white atrophy of right optic disc existed. The condition followed convulsions (post-partum), and was believed to be due to embolism or thrombosis of right Sylvian artery and of arteria centralis retinæ.

II.—*A Case of Glenard's Disease.* By J. P. Parkinson, M.D. *Exhibited October* 25, 1901.

A MARRIED woman æt. 40. She has had five children. She is said to have had no illnesses except pneumonia two years ago, and has never suffered from indigestion or abdominal pain or discomfort. She came to the hospital for symptoms due to stenosis and regurgitation of the mitral valve and cardiac dilatation.

The abdomen is very large and flabby from relaxation of the muscles of the abdominal wall. When lying on the back the lower limit of the liver can be felt a finger's breadth below the level of the umbilicus, and its dulness above extends to the costal margin, but in the erect position it falls to a much lower level. It can be freely moved about between the two hands. The spleen is also felt to be somewhat enlarged, prolapsed, and movable. The right kidney can be easily felt as a movable mass below the right limit of the liver, from whence it can be pushed back into the loin. The left kidney appears to be normal in position. The stomach

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DESCRIPTION OF PLATE XI.

To illustrate Dr. Seymour Taylor's case of Congenital Absence of both Clavicles.

seems to lie somewhat below its usual level, and to be slightly enlarged.

For a week in the middle of June the patient suffered from a constant pain in the right loin, which extended from thence to the anterior superior iliac spine, and during this period the urine, which previously had contained only a trace of albumen, diminished to half its usual amount, and contained two fifths of albumen. When the pain ceased, the albumen and, for a few days, the amount of urine increased to three or four pints daily. This temporary interference with the functions of the kidney was the only symptom referable to the large amount of visceroptosis present.

It is hardly necessary to add anything to the above except to mention that the increased weight of the organs from the congestion dependent upon the heart disease was a probable factor in the causation of the visceroptosis, and to say that the latter name, by suggesting a condition rather than a disease, perhaps fits the case better than the term "Glenard's disease," as the absence of symptoms was a remarkable feature in the case, which in as advanced types as the above usually causes some abdominal discomfort, and frequently hypochondriasis.

III.—*Case of Congenital Absence of both Clavicles.* By Seymour Taylor, M.D. *Exhibited October* 25, 1901.

C. W., male æt. 20, clerk. The family history cannot be obtained, hence the question of maternal impression cannot be answered. No history, however, of trauma or of fracture. There are some suspicious signs of hereditary specific disease. Both clavicles are incompletely developed. On each side the sternal ends, with the attachments of the sterno-mastoid muscles thereto, can be felt, and there would appear to be some attempt at development towards the acromial end. The intervening space is occupied by what would appear to be fibrous structure, possibly representing the costo-coracoid membrane. The upper limbs are not impeded in their various movements; indeed, the patient had no previous knowledge of his defects. On the other hand,

he can throw a ball and play cricket as well as most youths. Both shoulders can be so far approximated to the middle line that the eminences of the deltoid muscles can be made to touch each other. The condition thus approaches that of the Carnivora.

On each side, the sternal epiphysis bears no attachment of any fibres of the sterno-mastoid muscle, but there is a well-developed band of muscular fibres springing from the manubrium.

Attached, apparently, to the lower aspect of the rudimentary sternal end is a fibrous cord mentioned above, which, passing outwards towards the acromion, probably represents either the costo-coracoid membrane or the fibrous streak which is found in the Carnivora between the trapezius and the deltoid muscles, and which is doubtless the homologue of the clavicle in man.

IV.—*A Case of Hydrops Articuli in the Knee-joint of an Old Man who is the subject of Well-marked Osteo-arthritis.* By F. C. Wallis, B.C. *Exhibited October* 25, 1901.

THE ligamentum patellæ has been ruptured; previous to that the patella had been twice fractured; it can now be felt some distance up the thigh. In spite of this condition the man is able to get about with the aid of a stick. The large fluid swelling in front of the joint has been aspirated three times, and pressure has been applied. This has had no effect in reducing the fluid, and I have ceased to trouble about it. The comparative ease with which the man gets about with so disorganised a joint is unusual, and probably the presence of the fluid in the joint is a help in this respect.

V.—*A Case of Acute Intussusception in a child æt. 7 months (Ileo-cæcal variety) successfully treated by operation.* By F. C. Wallis, B.C. *Exhibited October* 25, 1901.

THIS patient was admitted to the Metropolitan Hospital on July 21 with somewhat obscure symptoms of intestinal obstruction. The operation was performed thirty-three hours after the first onset.

An incision in the upper part of the right semilunar line was made, and the abdomen opened. The intussusception, which occupied the situation of the hepatic flexure of the colon, was drawn out of the incision, and by gentle traction and pressure it was gradually reduced.

The intussuscepted gut was seven inches long, quite collapsed, somewhat congested, and thickened.

After reduction the intestine was returned to the abdomen, and the abdominal incisions closed by one row of catgut sutures. The operation lasted twenty minutes. The bowels acted next day.

Recovery was uninterrupted except that the baby was unable to digest any peptonised or other milk, but when the mother recommenced to feed it this trouble ceased.

The child was discharged on August 8 quite well.

VI.—*Case of Leontiasis Ossea, ? "Henpuye."* By H. Betham Robinson, M.S. *Exhibited October* 25, 1901.

T. P., male æt. 26.

Very hard, almost symmetrical swellings on the nasal bones and adjacent parts of superior maxilla, skin not involved. The nasal chambers are almost completely blocked, the turbinates being pushed towards the septum.

The swelling first appeared on the left side in 1897, and progressed slowly. No pain, no watering of the eyes, no history of syphilis.

He has been in South Africa since the lesions appeared.

The remarkable feature of the case is its strong likeness to "henpuye" or "dog-nose" of Cape Coast Colony.

VII.—*Pulsating Tumour of the Sacrum.* By CHARTERS J. SYMONDS, M.S. *Exhibited October* 25, 1901.

THE patient, a man æt. 65, came to Guy's Hospital complaining of pain in the sacrum. On rectal examination a pulsating tumour can be felt high up, covering the whole width of the sacrum, and reaching rather further to the right. The pulsation is controlled by compression of the aorta. Through the sacrum a loud systolic bruit can be heard. The patient has also outward displacement of the left eyeball, due to a formation (growth?) in the inner side. From the nose he had removed a year or more ago a polypus. It is suggested that pulsating tumour is a growth secondary to that in the ethmoid.

The patient pressed for some relief to the nasal obstruction, and so was put under chloroform, partly with a view to examining the pelvis more fully and at the same time for removing the more obvious polypus. No growth could be felt from above and no further mass by the rectum. On removing the polypus with a pair of forceps, a most furious hæmorrhage ensued, so that within a minute he must have lost the best part of a pint of blood; it was controlled by firm plugging with gauze. Very soon afterwards the left eyeball protruded to a much greater extent, and a marked bruit could be heard over the frontal bone, the appearances closely resembling those of arterio-venous aneurysm. As it was feared, symptoms of meningitis developed, and the patient died in a few days.

The *post-mortem* was made by Dr. Fawcett, who has kindly provided the following notes:

Geo. W., æt. 63 years. November 9, 1901, *post-mortem* six hours after death.

Nose: the upper portion of the left nostril was filled with a soft, rather gelatinous growth, invading the ethmoid and infiltrating its substance, both on the orbital and nasal aspects. The cribriform plate was softened by the growth, but the upper cranial surface was smooth and apparently covered by a layer of the dura mater; this was readily separated on taking hold of it with a pair of forceps.

Basal suppurative meningitis was present.

Supra-renals: left, softened and its interior excavated.

It did not contain any secondary deposits. Right, enlarged by a mass of soft white growth involving the whole of the organ, except for a small portion of its upper edge, of about 0·5 cm. in depth. The outline of the supra-renal was maintained, the growth being completely encapsuled. It measured 4·5 by 2·5 cm.

Kidneys: 736 grammes, considerably enlarged. Right, studded all over with rounded deposits of a white growth similar to that in the supra-renal; the largest of the deposits measured about 1 cm. across. Left, the upper half consisted of a mass of white growth, and at the lower margin of it was an area in which colloid degeneration had taken place. In the lower half of the organ were several secondary deposits like those in the right kidney.

Gluteal region and pelvis: *post mortem* there was no sign of any gluteal or sacral tumour when the body was examined from the outer surface. The gluteal muscles were cut through and dissected off, and no trace of any tumour was to be seen, either in this region or over the great sciatic notch. The external surface of the ilium appeared quite intact and of normal outline. On opening the abdomen and exploring the pelvis, no trace of any tumour could at first be found or made out. The large vessels and nerves were not involved, and it was not until the whole of the contents of the pelvis were cleared out that any trace of the tumour was detected. A section through the ilium at the level of the top of the great sciatic notch showed the bones to be perfectly normal. The growth occupied the body and lateral portions of the sacrum, spreading on the right side on to and into that portion of the ilium joining the upper part of the sacrum. The whole of the front of the sacrum had disappeared. There was no trace of any of the bodies, or of the anterior sacral foramina. All that remained was a shell of the sacrum formed of the posterior portion of the bone, which on its anterior surface was smoothed and grooved, forming a canal containing a solid mass of growth. The growth extended the whole length of sacrum, and over the coccyx was a projecting nodule of it.

The whole of the sacrum left was ankylosed together, and to it the articulating portion of the ilium. At the upper portion of the sacrum the growth had invaded the ilium for a distance of 5 cm., and protruded as a lobulated mass on the inner surface. The bone where invaded by the growth was so altered in its outlines that I could not definitely make

out its normal limits. Upwards the growth extended into the lower portion of the lumbar region, it having here also excavated for itself a groove similar to that in the sacrum.

The terminal portion of the cauda equina was involved. The growth in the groove of the sacrum measured 3·125 cm. deep by 3·75 broad.

There was no sign of any tumour externally, nor any growth protruding down over the great sciatic notch. Microscopically the growth was a small round-celled sarcoma.

I thought the nose was probably the primary growth, but that in the right supra-renal was very strikingly encapsuled, and did not invade the kidney in direct relation with it.

VIII.—*Syphilitic Myositis of the Extensor Cruris.* By CHARTERS J. SYMONDS, M.S. *Exhibited October* 25, 1901.

A MAN exhibited last session. He is again exhibited in fulfilment of a request made at that time. Doubt was expressed as to the nature of the case, some thinking the condition to be a new growth. In the interval rapid improvement has taken place, preceded by involvement of the skin. The patient was unable to submit to the treatment of rest, and for a time was without medicinal treatment. When he returned there was effusion into the knee, and signs of advancing disease, and the skin was broken in several places. Large doses of iodide produced rapid improvement, so that the diagnosis can no longer be doubtful.

IX.—*Arthropathy in a Case of Bronchiectasis.* Shown by H. E. SYMES THOMPSON (for PERCY KIDD, M.D.). *Exhibited October* 25, 1901.

ADA W., æt. 26, single. Symptoms of chronic bronchitis began about eighteen months ago, the sputum being offensive from the first. Twelve months ago the sputum became more offensive and more copious, and about this time

DESCRIPTION OF PLATE XII.

To illustrate Dr. Seymour Taylor's case of Bilateral Facial Palsy.

the ankle-joints first became affected. Subsequently the wrists and knees were involved.

Present state of joints.—Both wrists swollen, stiff, and tender; no creaking. Fingers generally enlarged, tips clubbed. Interossei and forearm muscles considerably wasted. Knees slightly swollen, and contain a little fluid. Ankles enlarged but not tender at present. Muscles of legs, especially extensors, wasted. Feet in the position of talipes equinus, but ankles can be dorsiflexed. Skiagraphs show no definite thickening of bones.

Physical signs of bronchiectasis well marked at left base. Sputum, formerly very offensive, has now no fœtid odour, though the quantity has not greatly diminished.

X.—*A Case of Bilateral Facial Palsy.* By Seymour Taylor, M.D. *Exhibited January* 24, 1902.

C. R. B., æt. 15, schoolboy.

He is stated to have had "paralysis of the legs and arms at five," from which he recovered.

His mother states that he has never been able "to move his face," and that, when he cried as an infant, she would not have known about it except for his open mouth and the noise that he made.

There is no history of delivery by forceps.

There is no history of otorrhœa.

He has had two attacks of rheumatic fever, viz. at ten and at fourteen.

He was admitted to the West London Hospital for consequent valvular disease of the heart.

There is almost complete bilateral facial palsy; but there would appear to be slight voluntary power in the risorius and the depressor anguli oris, and also in a few fibres of the orbicularis oris muscles. Otherwise the face is mask-like in its want of expression. The palpebral fissure is never completely closed. The mouth is always more or less gaping.

He talks readily, and can easily be understood by those who are accustomed to his manner of speech. But all labial and explosive consonants are pronounced by the aid of the tongue, so that "apple" is turned into "attle," "pear" becomes "tear," and so on.

He has some difficulty in the prehension of food. There is some movement in the soft palate during the pronunciation of vowels.

He has no other palsies, and no sign of degenerative changes in any other nerve.

The exact seat of the lesion has not yet been determined by complete electrical reactions, but the probability is, so far as we have been able to investigate, that the lesion is a peripheral one.

XI.—*Case of Dislocation of Navicular.* By G. R. Turner. *Exhibited January* 24, 1902.

JOHN L., æt. 32, farrier.

The patient was riding when his horse stumbled and fell with him, crushing his left foot; the man extricated himself, and in getting on to his feet twisted his foot inwards. After this he could not put his foot to the ground.

When he came up to the hospital the general appearance of the foot was like that of flat-foot.

There was a prominent rounded swelling just below and in front of the internal malleolus of the left leg. This swelling was quite easy to reduce by inserting the foot and pressing the swelling outwards, but it returned when the pressure was relaxed.

There was rather a marked hollow below the external malleolus, and decided abnormal mobility at the mid-tarsal joint.

The foot was examined under the X rays with a screen, and the scaphoid was seen to be dislocated inwards (see skiagram).

The foot was put up in a thickly padded internal splint so as to maintain it in a position of inversion, but it was found impossible to keep the dislocation thoroughly reduced.

Massage and movement were commenced after the first five days, and the patient got up and walked about with crutches in two weeks.

He left the hospital, walking without crutches, on November 30th.

DESCRIPTION OF PLATE XIII.

To illustrate Mr. G. R. Turner's case of Dislocation of Navicular.

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XII.—*Bilateral Cervical Ribs, and an Abnormal Bony Mass at the First Chondro-sternal Junction.* By Lawrie McGavin. *Exhibited January* 24, 1902.

GERTRUDE H., æt. 18. Patient first noticed the mass under the left clavicle six months ago; it had given rise to no inconvenience except that from time to time she had been troubled with aching pains in this region. On examining her the condition of her neck was accidentally discovered. She has lately complained of slight numbness and tingling over the dorsum of the first and second metacarpal bones of both hands. In addition to this she has occasionally experienced shooting pain in the area of distribution of the superficial branches of the cervical plexus, especially of the great occipital and supra-acromial divisions. The symptoms are more marked on the left than on the right side.

The skiagram shows the position of the supernumerary ribs; that on the right side is short and straight, and ends over the centre of the first rib, springing from the transverse process of the seventh cervical vertebra. That on the left is curved, and, springing also from the same point, appears to end much nearer to the inner end of the clavicle. It will be seen that the sternal extremities of the first ribs are markedly different in shape, that on the right being broad and thick, while that on the left is thin and indistinct.

At the upper margin of the skiagram is seen an indistinct nodular bony mass, apparently in connection with the transverse processes of the fourth and fifth cervical vertebræ.

XIII.—*A Case of Abnormal Curvature of the Spine determined by Shortening of the Left Rectus Abdominis Muscle.* By J. Jackson Clarke, M.B. *Exhibited January* 24, 1902.

A. B. was first brought to me in December, 1900. She was then nine years old, and she had a pronounced C-shaped total kyphosis, associated with slight dorsal scoliosis,

the convexity of the curve being to the right. The patient's breathing was purely abdominal, and the abnormal curves in the spine were fixed, not disappearing either when the patient lay down or when manual pressure was employed. On attempting to straighten the spine, I observed that the lower portion of the thoracic wall on the left side was dragged upon, and that the left rectus muscle was tense and hard in every position of the body, and that it was less resistent when the patient was sitting with the body bent forward than it was when she lay down on the back. On inquiry I found that when the patient was six months old her mother had noticed that the right side of the abdomen was more prominent than the left. The case is probably akin to the common form of wry-neck, due to organic shortening (contracture) of one sterno-mastoid in infants.

The treatment has been instrumental and gymnastic. Improvement has been thus obtained through the stretching of the shortened rectus; it remains to be decided whether any marked gain would result from elongation of the left rectus by a simple plastic operation.

XIV.—*Arthropathy of Shoulder-joint in a Case of Syringomyelia.* By W. D. Harmer, M.C. (introduced by Anthony Bowlby). *Exhibited January* 24, 1902.

T. P., male æt. 20 years, hawker.

Injury to left shoulder twelve months previously, followed by swelling (? ruptured muscle), which gradually subsided.

Eight months ago painless swelling of same joint, commencing suddenly without injury.

Two months' grating and "shifting of bone." "Back curved" last three or four years (?). Family history not important. No previous illness except varicella. No history of syphilis. Generally has boils on some part of body.

DESCRIPTION OF PLATE XIV.

To illustrate Mr. Harmer's case of Arthropathy of Shoulder-joint in a case of Syringomyelia.

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DESCRIPTION OF PLATE XV.

To illustrate Mr. Harmer's case of Arthropathy of Shoulder-joint.

Short, strongly built patient; voice always husky. No muscular wasting discovered. Grip in left hand a little weaker. Left shoulder-joint uniformly enlarged (18 inches by 15 Rt.), painless. Movement free as high as the level of acromion process, but attended with marked grating and luxation.

Head of humerus eroded (*vide* skiagram). When arms are hanging there is an inch of lengthening of left due to laxity of ligaments. Well-marked kyphosis and lateral curvature with convexity to the left (*vide* photograph). Not tender on pressure. Secretion of sweat excessive on face. Numerous painless ulcers on thumb and fingers of left hand, commencing as bullæ. Complete thermo-anæsthesia from about fifth cervical to third lumbar spines, including arms. Analgesia marked only over front and lateral aspect of joint; slight in left hand. Tactile sense normal. No loss of muscular pressure or localisation senses. Knee-jerks marked. No ankle-clonus. No ocular changes.

XV.—*A Case illustrating a New Method of treating by Muscle-grafting Old-standing Paralysis of the Upper Root of the Brachial Plexus (Erb-Duchenne Type).*

By A. H. Tubby, M.S., and Purves Stewart, M.D.

Exhibited January 24, 1902.

THE following case is of considerable interest, as it shows the results of an attempt to treat an often incurable affection, and to render a useless limb of service for some of the ordinary duties of life. The notes on the case are as follows:

The patient, Thomas N., æt. 56, is by occupation a carman, and he sought admission to Westminster Hospital on account of loss of power in the left arm, which entirely precluded him from following his vocation. He is a big and stout man, inclining to obesity, but he states that his general health has been very good, and he has never suffered from any illness he can remember, and is moderate in taking alcohol.

On February 11, 1901, he was admitted to Westminster Hospital under the care of Dr. Murrell, and said that sixteen months ago he fell off his van—a distance of about nine feet—and struck the left shoulder and left side of his head, and was stunned for over an hour. On regaining consciousness he found himself unable to lift the left arm, and there was a large amount of swelling around the shoulder, accompanied by some bruising. A week after the accident he went to the Temperance Hospital, which he attended for six months, and then to the Hospital for Paralysis in Queen Square for another six months, but without gaining any benefit. The treatment consisted in external applications and the use of electricity. There is no evidence of any fracture or dislocation.

When he came into Westminster Hospital there was found to be wasting of the deltoid, infraspinatus, biceps, brachialis anticus, and supinator longus on the left side, and he was quite unable to abduct the shoulder or to flex the elbow. There was entire loss of electrical excitability in these muscles, and it was noted that there was anæsthesia along the outer side of the upper arm and forearm in the areas of skin supplied by the circumflex and musculo-cutaneous nerves.

He was treated by application of the faradic current and hypodermic injections of strychnine, and it was found on April 2, 1901, that the anæsthesia had disappeared, but the motor paralysis remained as before.

On April 26 an examination of his condition by Dr. Purves Stewart gave the following results:

The movements of the scapula are seen to be normal, and sensation is good. He can rotate the humerus inwards but not outwards, nor can he abduct the humerus. The action of the pectoralis major and minor is strong. He makes feeble attempts to flex the elbow, and when doing so the forearm is first pronated and the wrist is then strongly extended to the radial side, and the extensor carpi radialis longior is felt to contract. There is good extension of the elbow, with normal pronation and supination of the forearm, and the movements of the digits are good. The deltoid, infraspinatus, biceps, brachialis anticus, and supinator longus do not react either to faradism or galvanism, but the pectoralis, trapezius, and muscles of the forearm do so.

Dr. Purves Stewart, who had seen the case several times with Dr. Murrell, then consulted Mr. Tubby as to whether

anything could be done surgically, and muscle-grafting was suggested by Mr. Tubby. It was proposed to do this in two stages: firstly, to attempt to obtain flexion power at the elbow by transferring part of the triceps into the biceps; and secondly, to restore the abduction power of the humerus by grafting part of the pectoralis major into the deltoid, turning the reinforcing part of the pectoralis major over the acromion. The patient's cardiac condition was not such as (in the opinion of Dr. Bourns, the anæsthetist to the hospital) to render it safe to give a general anæsthesia, so it was decided, at Dr. Bourns' suggestion, to do the operations under eucaine.

On June 10, 1901, after a 4 per cent. solution of eucaine had been injected, an incision six inches long was made, beginning from the middle of the back of the upper arm, and curving downwards and forwards over the musculo-spiral groove. The musculo-spiral nerve was rapidly defined, and the outer border of the brachialis anticus. The biceps muscle was found to be atrophied, and a small portion was removed for microscopical examination. The external head of the triceps was then laid bare, and a strip, an inch and a half wide and one inch thick and from three to four inches in length, was made by cutting through the substance of the muscle vertically and parallel to its fibres, and then dividing the lower end of the strip some little distance above the olecranon. An aperture having been made through the middle of the biceps tendon, the lower end of the strip was carried across to beneath the biceps, made tense, and drawn through it, and secured with kangaroo-tendon sutures. The operation lasted thirty minutes, and the patient experienced very little pain.

The arm was put up with the elbow well flexed, and by June 28 the wound was completely healed.

On July 1 the second operation was undertaken. An incision eight to nine inches long was made, commencing just below the middle of the clavicle, then passing across the tip of the acromion and downwards over the deltoid for about four inches. The inner border of the deltoid was defined, and the division between the sternal and clavicular portions of the pectoralis major also defined. The clavicular part of the latter muscle was separated distally near the humerus, and the end was carried up over the acromion and then fixed into the deltoid. As it passed over the acromion the muscle was quilted down by kangaroo-tendon sutures.

Except for the collection of a little serum the wound did quite well.

The patient was afterwards transferred to a medical ward, where both galvanic and faradic currents were applied. At first there was total loss of excitability to both currents, but by October 10, 1901, galvanic excitability had returned in the grafted muscles. It was also found that the portion of the triceps transplanted to form a new biceps was acting satisfactorily, and the patient had acquired a good degree of flexion power, and there is no doubt that, so far as flexion is concerned, there has been a very great improvement. But although the transplanted portion of the pectoralis major can be felt to harden when attempts are made to abduct, yet the patient is unable to lift his arm away from the side without corresponding movement of the scapula.

On December 4 it was found that the faradic reaction could be demonstrated in both the transplanted muscles.

Remarks.—Although the results in this case are not quite perfect, yet there is no doubt that the patient's condition has been much improved by the operations, that he will be enabled to resume his occupation of carman, and that a useless limb has been transformed into a useful one. Unfortunately the case was not an entirely favourable one for this kind of treatment. The man's bulk and the fatty condition of his heart, which rendered a general anæsthetic unsafe, caused the operations to be done somewhat hurriedly under a local anæsthetic; while the size of the limb and its weight, with the great leverage involved, made it very difficult to obtain sufficient muscular power to secure quick and rapid movement.

It might have been thought advisable to transfer a portion of the trapezius instead of the pectoralis major, but this did not promise so well, because the trapezius is a thin muscular sheet unlike the thick pectoralis major; and, further, it was thought that, if the trapezius were interfered with, any attempts to abduct the arm by means of the transplanted portion would be associated with movements of the head towards the affected side.

Examinations of the portions of muscle removed showed that they were profoundly atrophied. The sarcolemma nuclei had undergone proliferation, the individual muscle-fibres had become smaller in diameter, and in many cases had lost their striation.

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DESCRIPTION OF PLATE XVI.

To illustrate Mr. Barnard's case of Coxa Vara.

XVI.—*A Case of Coxa Vara, probably due to Bending and Absorption of the Neck of the Femur from a Tubercular Deposit in it.* By HAROLD L. BARNARD, M.S. *Exhibited January* 24, 1902.

E. W., a little girl æt. 9, was brought to me for a limp and stiffness in the right hip. The movements of the hip were painless and free, with the exception of abduction and internal rotation, which were distinctly limited. No limitation of extension existed. The right leg was 1½ inches shorter than the left, and the right trochanter was well above Nélaton's line, and much more prominent than the left. Its circle of rotation and the distance from the symphysis pubis were also diminished. On palpation no tenderness or swelling was discovered. There was some wasting of the entire leg, but not more than could be accounted for by disuse.

The accompanying skiagram was taken, and left no doubt that the condition was one of coxa vara. It will be seen that the neck of the femur has not only been bent downwards, but also largely absorbed, so that the head is now planted nearly on the upper end of the shaft between the trochanters. The antero-posterior curve in the neck of the femur usually seen in cases of coxa vara is little marked.

The condition of her lungs was as follows :—At the right apex, over the situation of the upper lobe, and both in front and behind, impaired resonance was found on percussion, and some sharp crepitations were heard. There were no signs of a cavity or of any considerable consolidation. The child was, moreover, of the coarse strumous type, and had tubercular glands of the right neck, and a caseating gland in front of the right ear of the parotid set. The history of the child was interesting.

Some three or four years ago she attended the Royal Chest Hospital in City Road for some disease of the lungs. Eighteen months ago she was staying with her parents at Southend, when she complained suddenly that her right hip hurt her, and she could not walk. There is no history of a fall, but her mother made no inquiries at the time, and the child, who is rather dull, cannot be induced to remember anything about the occurrence at all. The mother took the child to a chemist, who said it was rheumatism and prescribed a liniment. For

three days the child could not walk, and was wheeled about in a perambulator. Then she apparently recovered entirely, and got about as usual. Soon after progressive disability began to appear in the hip, and became worse and worse, until the child was brought to my out-patients at the Metropolitan Hospital. The case is an obscure one, but the diagnosis appears to lie between—

(1) A localised tubercular deposit in the neck of the femur, not involving the synovial cavity of the joint, but so weakening the bone that it bent and shortened under the strain and weight of the child. The sudden onset may perhaps be explained by a spontaneous subperiosteal fracture determined by a tubercular focus.

(2) A fracture of the neck of the femur, with subsequent osteitis, softening, and bending, due to a forgotten fall or jar.

If the latter explanation be accepted, it is interesting to compare this case with that of a separated upper epiphysis of the femur shown by Mr. Jonathan Hutchinson, jun., at the same meeting. This separation occurred in a boy of seventeen years of age. Mr. Royal Whitman has shown that in trauma of the neck of the femur in children a fracture occurs because the epiphysis is not yet sufficient of an entity to be separated, and the neck is a weaker point in the bone than the epiphysial line. The typical age for separated epiphysis, Mr. Whitman contends, is sixteen to eighteen years. The child is being treated with a Thomas's knee-splint with extension, and a patten on the left boot. The parents will not consent to operation.

XVII.—*Compound Comminuted Fracture of the Right Patella: suture with kangaroo tendon: late suppuration: drainage of the knee-joint: bony union and good movement.* By HAROLD L. BARNARD, M.S. *Exhibited January* 24, 1902.

J. B., æt. 21, was a railway porter. He was crossing the line in a station, and seeing a train approaching he tried to scramble on to the platform. The engine, however, struck his right knee, and threw him across the platform.

When admitted to the London Hospital he had a wound

over his right patella 2 inches in length, and through this projected the lower fragment of his patella. The knee-joint was full of blood, which infiltrated the surrounding tissues and flowed freely from the wound. The knee and lower fragment were grimed with dirt.

Operation.—He was anæsthetised and the knee and projecting fragment carefully cleaned. Ragged, dirty tissue was cut away, and the edges of the wound resected. A flap was then turned up, and it was then found that the fragments had separated two or three inches, and that both were broken into several pieces, and the lower one split horizontally. The lateral patellar ligaments were rent back to the lateral ligaments. The subcutaneous tissue was infiltrated with blood-clot, and was half an inch thick.

The knee-joint was freely flushed out with sterile saline solution, and the lateral patellar ligaments were laced up with kangaroo tendon according to the method described by Stimson. The aponeurotic fringes were then cut away from the fragments, and these were drawn into close contact by a stout kangaroo tendon passed transversely across the tendon of the quadriceps and the ligamentum patellæ, and tied in front of the patella. The flap was sutured and a drainage-tube inserted in one angle. This tube was removed with all possible precaution on the fourth day, when only serum had escaped.

The temperature remained normal until the seventh day, when it rose to 101° F., and oscillated at about that level during the weeks of chronic suppuration which followed. The sepsis took the form of a chronic sloughing cellulitis, and nearly all the subcutaneous tissue came away. About fourteen days after the accident the knee-joint became swollen, and was apparently infected. Drainage-tubes were therefore inserted in front through the rents in the lateral patellar ligaments, which were reopened for this purpose, and behind the sartorius and biceps tendons, so as to drain the posterior pouches of the knee-joint separately, according to the method described by me in the *Practitioner* for October, 1901.

Clear serum escaped from each of the parts of the knee-joint incised, but there was no well-marked suppurative arthritis.

After the drainage of the joint the temperature slowly fell to normal, and the wounds became healthy and healed. As soon as the healing process was established, massage and passive movements were carried out with assiduous care by

Mrs. Palmer, masseuse to the London Hospital, to whom most of the success of this case is attributable.

The whole patella not yet united was wabbled on the condyles of the femur to prevent adhesions binding down the upper fragments. The upper fragments were then drawn firmly down, so as to relax the strands of kangaroo tendon, and the knee was lifted from the bed so as to flex the joint.

When shown at the Clinical Society, three months after the accident, the patella had united by bony union, and the patient could fully extend the knee-joint and flex it to a right angle. He could walk well and strongly, and had only a slight limp, and all his wounds had healed. This case illustrates the following points:

(1) The advantages of kangaroo tendon over silver wire, for had the latter been used it would almost certainly have had to be removed, and would probably have led to necrosis of the bones, which had been drilled, and wide gaping of the fragments. Moreover, out of the shattered fragments it would have been hard to say which should be wired together.

(2) The advantages of employing an efficient method of draining the knee-joint; that is to say, one which gives a free escape for pus from both posterior pouches of the joint.

(3) The inestimable advantages of intelligent massage and passive movement in such a case, according to the principles laid down by P. Bruns, Lucas-Champonnière, and Bennett.

XVIII.—*Traumatic Separation of the Epiphysis of the Head of the Femur demonstrated by Skiagraphy.* By J. Hutchinson, jun. *Exhibited January* 24, 1902.

THE real nature of this case would probably have been overlooked, and could not have been ascertained with certainty, but for the aid of the Röntgen rays. The patient was a lad æt. 17, of poor muscular and osseous development, who was admitted into the London Hospital on November 22, 1901. Whilst struggling with some boys he had fallen on to the ground, and it is probable that at the moment of the fall there had been forced extension and

DESCRIPTION OF PLATE XVII.

To illustrate Mr. Hutchinson's case of Traumatic Separation of the Epiphysis of the Head of the Femur.

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eversion of the right hip. When brought to the hospital he was unable to walk, and complained of intense pain on passive movement of the right thigh. The latter was everted just as in most cases of impacted fracture of the femoral neck, anterior dislocation, etc. There was no bruising over the trochanter, and no abnormal fulness in Scarpa's triangle. The right ilio-tibial band was slightly relaxed, but yet no shortening could be made out. Pressure on the trochanter region did not cause pain. The Röntgen rays showed very plainly a separation of the femoral head exactly at the epiphysial line, the epiphysis remaining in the acetabulum, and being only in contact with the femoral neck to about half the normal extent. Under an anæsthetic the limb could be freely moved in all directions, and a faint sort of crepitus was obtained. The eversion could easily be overcome, but when the extension was taken off the deformity at once recurred. The limb was put up on a Liston's long splint, and union occurred, less than half an inch shortening. For some obscure reason, however, the ultimate result has been considerable stiffness of the joint. Under anæsthesia this loss of mobility can be completely done away with, but it recurs in spite of every care. The lad is weak and the subject of genu valgum.

Until the introduction of the X rays, positive evidence as to the occurrence of traumatic separation of this epiphysis was singularly scanty. It had been proved to occur in lower animals, such as the horse, the pig, and the rabbit, but there was apparently only a single specimen of it obtained from the human subject. This specimen, described and figured by M. Bousseau in 1867 (*Bulletin de la Société Anatomique*, 1867, p. 283), has unfortunately been lost, but it is of interest to note that it was obtained from a patient nearly the same age as my own, *i. e.* fifteen years.

In the Jacksonian Prize Essay 'On Injuries to the Epiphyses' I have collected notes of twenty cases in which there was good reason to believe that in young subjects either separation of the epiphysial head or fracture through the neck had occurred. Nothing can be more conclusive than the evidence of Pl. XVII, and it will probably be found that the injury is by no means so uncommon as was at one time supposed, and that it is one cause for the development of unilateral coxa vara.

XIX.—*Major Dilatation of the Left Auricle.* By W. Ewart, M.D. *Exhibited January* 24, 1902.

XX.—*Case of Excision of the Cæcum.* By Arthur E. Barker. *Exhibited January* 24, 1902.

CASE 1.—W. B., æt. 30, was operated on for acute intestinal obstruction on May 7, 1899.

The enormously distended and hypertrophied small intestine was first incised, and five pints of liquid fæces were evacuated. The cause of the obstruction could then be made out to be a hard growth round the ileo-cæcal valve. The small gut, at about two feet from its termination, was therefore anastomosed laterally with the transverse colon. Six weeks after a fresh incision was made over the cæcum, and the latter was excised with some inches of the ascending colon and ileum. Beyond the presence of a slight fæcal fistula for some weeks, recovery was perfect. The patient is now in excellent health, without any evidence of recurrence, two years and eight months after operation. The growth was a typical carcinoma. (Vide *Clin. Soc. Trans.*, 1900, p. 29.)

XXI.—*A Case of Excision of Transverse Colon.* By Arthur E. Barker. *Exhibited January* 24, 1902.

CASE 2.—Mrs. M., æt. 58, was operated on for carcinoma of the transverse colon and chronic obstruction on October 25, 1900. She was suffering at the time from diabetes, with 6·6 per cent. of sugar in the urine, and had been for years. About four and a half to five inches of the transverse colon were removed, and end-to-end anastomosis followed. The dressings remained untouched for ten days. On their

removal a small fæcal fistula was found, and discharged for some weeks with one of the suture threads, probably omental. Recovery was otherwise uneventful. The growth was typical carcinoma. (Vide *Medico-Chirurgical Transactions,* 1901.)

The patient has been in first-rate health ever since, and shows no evidence of recurrence.

XXII.—*A Case of Compound Fracture of the Tibia, the Superjacent Tissues being riddled with Suppurating Gummata.* By F. C. WALLIS, B.C. *Exhibited January* 24, 1902.

THE patient was admitted to the Metropolitan Hospital with a bad compound fracture of the right tibia. The fracture and the skin lesions were clean transverse wounds. Above and around were numerous sloughing suppurating gummata. There were many pigmented scars, and the skin over the whole of this part of the leg was thickened and infiltrated, and of a purple colour. The fractured ends of the tibia were freely exposed through the gaping skin wound.

Owing to the condition of the parts, it was determined that the fracture should not be covered in any way. It was accordingly placed in a back splint with a foot-piece, slung in a cradle, and exposed to the view. The only application was iodoform powder. As the pus accumulated in the various ulcers it was wiped out with wool and fresh powder applied. Potassium iodide was given internally.

The leg has united very well, in quite good position, and the various gummata are nearly healed.

XXIII.—*A Case of Ataxic Paraplegia.* By NORMAN DALTON, M.D. *Exhibited January* 24, 1902.

CHAS. G., æt. 52, contracted syphilis two years ago. On admission had ulcers on the legs, which are nearly healed. As far as can be made out his present symptoms

have been coming on about one year. His mind is sound, but he is emotional and excitable, and it is quite possible that some of his statements are incorrect.

His symptoms are partly those of locomotor ataxy and partly those of lateral sclerosis. The eyes and the upper limbs are normal. As a rule he stands fairly steadily with his eyes shut. He complains of "precipitate" micturition and defecation. Sometimes the action of the bladder is "stammering." In "walking" he shoots his legs forwards and rather too much outwards, and he brings his foot down forcibly on the heel as in ataxia; but he does not lift the knees, and the movements of the legs are not free, but appear to be affected by a certain amount of stiffness. In particular the toes are turned up. The muscles of the legs and thighs are not rigid, but rather flaccid. Their electrical reactions are normal. The "knee-jerks" are greatly exaggerated, particularly on the left side, and ankle-clonus can be obtained always in the left foot, but not always in the right. The plantar reflex is of the extensor type. There is no delayed sensation. He has complained of pains in the left leg, shooting from the hip to the foot, sometimes accompanied by twitching movements. In the right lower limb, as high as the crest of the ilium (back and front), there is loss of painful sensation, and of sensations of heat and cold. The sense of touch is retained, and the sole of the foot is very sensitive to touch. At one time the left lower limb was imperfectly sensitive to touch, but this has passed off. However, it is not easy to be positive about any of his sensory phenomena, except that the loss of painful sensation in the right lower limb appears to be constant. There is no loss of the sense of position of the lower limbs. There have been no definite crises, but he has had pains in the hypogastrium.

XXIV.—*A Case of Congenital Spinal Degeneration with Symmetrical Arthropathy.* By W. Essex Wynter, M.D. *Exhibited January* 24, 1902.

WILLIAM B., æt. 17, pony driver in a coal-pit.

Family history.—Father and mother living; former suffers with rheumatism. Three brothers and two sisters in

Jas. Mackenzie Davidson.

PALMAR ASPECT.

These when viewed in the Stereoscope (by a person possessing bin-ocular vision of course), will give an image of the hand in relief viewed from the *palmar* aspect. The semilunar bone will be seen dislocated forwards into the palm.

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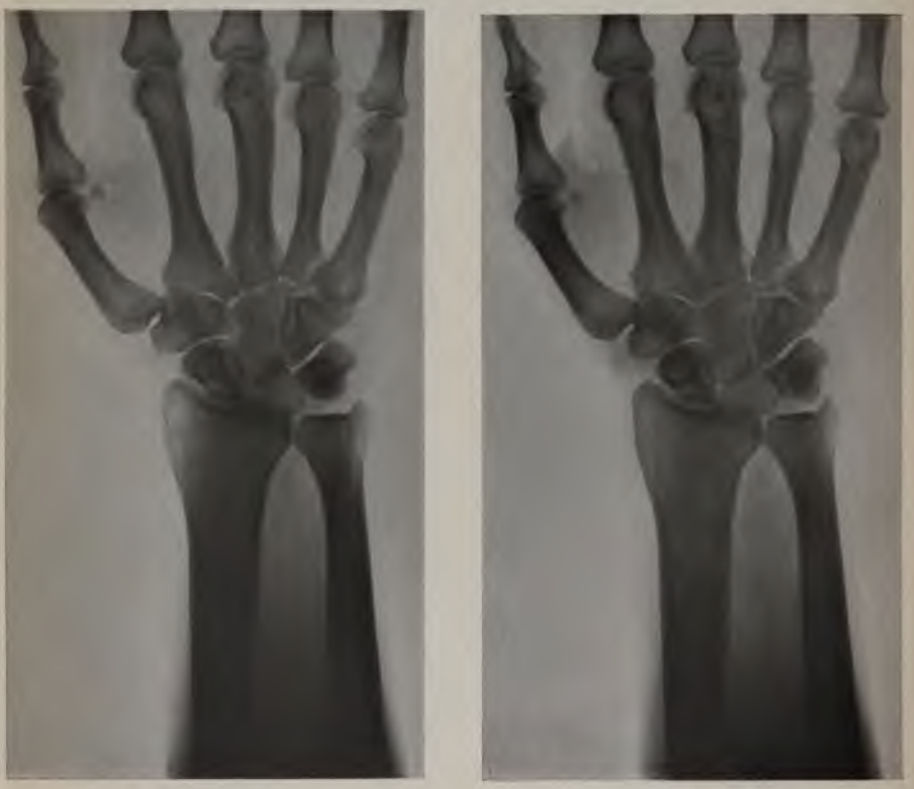
DESCRIPTION OF PLATE XVIII.

To illustrate Mr. Marsh's case of Dislocation of the Semilunar Bone.



JAS. MACKENZIE DAVIDSON.

DORSAL ASPECT.

When these are viewed with an ordinary lenticular or Brewster's Stereoscope—a realistic image in true Stereoscopic relief is seen. The observer sees the hand from the *back* and the semilunar bone is distinctly seen dislocated forwards.

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DESCRIPTION OF PLATE XIX.

To illustrate Mr. Marsh's case of Dislocation of the Semilunar Bone.

good health; two brothers dead, cause unknown; no similar disease known in family.

Previous history.—Patient was kicked over the left eye by a horse at fourteen.

Present condition was noticed two years ago, commencing without pain or any acute phase. At first difficulty was experienced in walking, then it was noticed that the right hip was stiff. Dr. Tweedie, of Nottingham, noted that there was then no deformity or shortening, but Thomas's splint was applied. In May, 1901, the left hip was similarly affected, and by June there appeared to be no movement at all in either.

Admitted December 9, 1901. Fairly developed and well nourished. Organs appear healthy. Both hips are completely fixed in a fully extended position, so that the patient is unable to sit up in bed or in a chair. There is some enlargement of the right trochanter. Manipulation elicits no tenderness. Measurement from the anterior superior spine to the internal malleolus 33½ inches on either side. Both trochanters are below Nélaton's line. Bryant's line—left 2⅜, right 2⅝ inches.

There is no wasting or loss of sensation or muscular power.

Electrical reactions are normal. Reflexes uniformly exaggerated.

The patient stands in a peculiar attitude, with knees slightly flexed, and the spine curved forward. He walks by rotating the pelvis and spine.

After fourteen days' treatment by iodide of mercury there is some movement in the left hip; urine normal; temperature normal.

The hip-joints were twice flexed under chloroform, only slight resistance being encountered. On both occasions rigidity recurred at once.

XXV.—*A Case of Dislocation of the Semilunar Bone.* By Howard Marsh. *Exhibited February* 28, 1902.

CASE.—*Complete dislocation of the semilunar bone of the right carpus forwards, with fracture of the anterior border of the articular surface of the radius; removal of the semilunar*

bone and of the callus on the radius; power of flexing the fingers regained.—W. T., æt. 27, serving in South Africa, was thrown from his horse, and received severe injury of his right wrist. On his return to England he had little power of flexing his fingers, and the effort was attended with pain. There was a hollow at the back of the carpus in the situation of the semilunar bone, and this bone could apparently be felt lying on the anterior surface of the radius, the flexor tendons being stretched over it. A skiagram, taken by Mr. Mackenzie Davidson, confirmed this diagnosis. A few days later a vertical incision just above the wrist was made, and the flexor tendons separated. The semilunar bone was readily exposed, but its extraction was attended with some difficulty, for it was partly embedded in callus formed as the result of a fracture splitting off a portion of the articular surface of the radius. This callus was freely removed, so that the flat surface of the radius was restored. The wound healed by primary union, and the full power of flexing the fingers has been regained. For some weeks the index and middle fingers remained "asleep," but this defect is by degrees passing off. The displacement was extremely well shown in Mr. Mackenzie Davidson's skiagram (exhibited), but the fracture of the radius, which was of limited extent, did not appear.

XXVI.—*Demonstration of a new form of Æsthesiometer for estimating the Degree of Acuteness of Tactile Sense at different parts of the Skin.* By GRAHAM BROWN, M.D. (introduced by the PRESIDENT). *Exhibited February* 28, 1902.

THE instrument consists essentially of a smooth surface, which can be made rough to any desired degree by means of adjustable projections from this surface. It is specially suitable for clinical work in cases in which tactile sensation is impaired.

XXVII.—*Case illustrating the Treatment of Bronchial Dilatation and Bronchial Catarrh by Posture and Respiratory Exercises.* By W. EWART, M.D. *Exhibited February* 28, 1902.

THE method consists in *keeping the foot of the bed raised,* sometimes to a considerable height, whilst the shoulders are kept low, only the head resting on the pillow. The first case, one of severe bronchiectasis, in which it occurred to me to carry out this indication has been published; others, including some cases of phthisis, have been under treatment. The method, however, had been previously thought of and described by Quincke.

Posture such as that described, particularly if kept up continuously, fulfils the first essential by saving the tubes from accumulation and from irritation, and may thus in advanced cases afford protection against serious complications, whilst freeing the patient from the evils of cough strain. Any other treatment that may be adopted simultaneously, is in this way given a considerable advantage.

The respiratory exercises, the other part of the treatment which I recommend, are intended to improve the ventilation and the circulation in the damaged lung, and to reclaim for pulmonary tissue the bronchial space vacated by the previously retained secretions.

Case of chronic pulmonary catarrh and slight bronchiectasis in a kyphotic subject.—The physical signs of bronchiectasis are not obtained definitely because of the thoracic deformity, but the fingers are clubbed, and the history of occasional large gushes of expectoration, particularly during the last few months, suggests the existence of dilatation.

J. G., hawker, æt. 23, admitted to St. George's Hospital February 11 ('Med. Reg.' 248, 1902), had always had a tendency to catarrh during the winter, but had been much worse during the last ten years. He was recently in bed for three weeks, with severe suffocative catarrh and copious expectoration, orthopnœa, and straining cough. On admission he was dusky and almost cyanotic, with imperfect resonance at the back of both lungs. He improved under expectorant treatment, but on February 15, to relieve the cough, eight-inch blocks were put under the foot of his bed.

This had the immediate effect of relieving all struggle of cough and of facilitating the outflow of expectoration. The temperature ceased to be elevated the next day.

XXVIII.—*A Case of Locomotor Ataxia (?) (Charcot's Disease of the Shoulder-joint ?).* By JOHN R. LUNN. *Exhibited February* 28, 1902.

HENRY H., æt. 65 (?).

The patient is very deaf and weak-minded, consequently it has been difficult to obtain any sort of history or present condition from him.

No paralysis of muscles, walks fairly well, no ataxia, can stand with his eyes shut and feet together. No tremors of extremities. Patellar reflexes absent on both sides. Plantar reflexes increased. Left pupil small, reacts to light; the right pupil dilated, and does not react to light. Sometimes there is incontinence of fæces and urine.

The left shoulder-joint flail-like, very movable, and when moved does not complain of pain. The muscles of the upper arm (left) are not markedly wasted. Slight grating in the joint. The last phalanx of the right thumb atrophied, and can be hyper-extended on the proximal phalanx, and most of the joints are deformed in the right hand in the same way, which the patient attributes to an accident.

The patient seems to have improved somewhat under antisyphilitic treatment.

XXIX.—*A Case of Polyarthritis in a man æt.* 30 (*cause ?*). By JOHN R. LUNN. *Exhibited February* 28, 1902.

THOMAS W., æt. 30, a carman.

Family history good. No gout or rheumatism in the family; never had gonorrhœa or syphilis.

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DESCRIPTION OF PLATE XX.

To illustrate Mr. Lunn's case of Osteitis Deformans.

Three years ago noticed pain in the left ankle, which began to swell, and remained swollen for twelve months, and then the right began in exactly the same manner. About eleven months later both wrist-joints began to swell, and then the right knee. At present there is a good deal of thickening round both ankle-joints, and fluctuation can be obtained; they are tender, but not red or hot. There is also well-marked flat-foot (bilateral) and swelling in the right knee-joint; muscles of the calf are much wasted. Both wrist-joints are swollen and painful; also the metacarpo-phalangeal joints of the first, second, and third digits on the right side, and first and second on the left hand. Thenar eminence on the right side is wasted, but not on the left. No headache or vertigo. The pupils act to light and accommodation. The knee-jerks are brisk on the left side, not obtainable on the right, apparently owing to the swelling of the knee-joint. Never had any rise of temperature. The patient has not improved under any treatment.

XXX.—*Osteitis Deformans, with Skiagram.* By JOHN R. LUNN. *Exhibited February* 28, 1902.

WILLIAM W., æt. 72. Family history good. Rheumatic fever when twenty years of age. Right clavicle broken in 1888; no history of syphilis or gout.

Present condition.—The patient carries his head forward from his shoulders, and stoops in walking. His gait is shuffling and tottery. There is general hyperalgesia of the skin. No anæsthesia. Knee-jerks active, more especially the right. He exhibits stiffness when moving his head. There is no curvature of the spine when resting in bed. All his articulations appear to act well and to be free from any creaking. Most of his teeth are lost or decayed. His nails are brittle and longitudinally grooved. There is some fulness about his ankle-joints. Thickening of articular ends of middle phalanges, also terminal ends of proximal phalanges. No apparent thickening or curvature of the arm bones. The spine of the scapula is thickened. Both clavicles appear thicker than normal, especially the right. The cranium looks bony, and seems too large for the face. The occipital bone

feels thickened, also the upper angle of the frontal bone and the temporal ridges. The innominate bones show thickening of their upper bodies, and their curvatures seem somewhat exaggerated. The right femur is much curved, and feels massive to touch; the centre of the curve corresponds to the middle third of the shaft; the internal condyle is prominent. The left femur shows a similar condition, but in a slighter degree. Both tibiæ show marked curving forwards about their middle third.

XXXI.—*Excision of the Lower End of the Ulna for an old Colles' Fracture.* By CUTHBERT T. WALLACE. *Exhibited February* 28, 1902.

G. W., male æt. 55, was admitted in August, 1899, for loss of power in the hand in consequence of a Colles' fracture, which he had received some time previously. The grasp of the hand was very weak, and incapacitated him from doing any useful work. There was much abduction of the hand, and flexion was extremely limited, owing to the lower end of the ulna overlapping the carpus. About three quarters of an inch of the ulna was removed through an incision on the inner side of the wrist.

The skiagrams show the condition before and after operation.

Power has returned, so that the patient can follow his occupation.

XXXII.—*A Case of Sarcoma of the Synovial Membrane of the Ankle-joint.* By G. R. TURNER. *Exhibited February* 28, 1902.

THE patient is a man æt. 28.

History.—Two months ago patient knocked his foot against a brass screw. Five weeks ago first noticed swelling in front of right ankle. This has increased slightly, and makes the foot "seem weak." No previous illness. No history of tubercle.

DESCRIPTION OF PLATE XXI.

To illustrate Mr. Wallace's case of Excision of the Lower End of the Ulna.

FIG. 1.

FIG. 2.

FIG. 3.

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WRONG

On examination.—In front of the right ankle, just anterior to the external malleolus, is a hard, rounded swelling, not reducible into the joint, about one and three quarters by one and a half inches in diameter. A cutaneous nerve is stretched tightly over the swelling, which latter does not seem obviously connected with the tendons.

January 16.—Mr. Turner cut down upon the tumour, which was found to be a solid growth springing from the synovial membrane of the joint, the latter being opened as the tumour was removed.

The tumour was flat on its joint surface, with numerous outgrowths and excrescences in front and on the edges, in places being very dark brown in colour.

Temperature normal except day after operation.

Dr. Rolleston reports "a mixed-celled sarcoma containing a few giant-cells. There is fibrosis and commencing chondrification. It is not a myeloma, but a mixed-celled sarcoma."

Discharged February 3.

XXXIII.—*Union by Secondary Suture of an Ilio-lumbar Incision after the Simultaneous Evacuation of an Abscess and the Excision of the Diseased Appendix, the abscess having recurred for the fourth time.* By W. G. Spencer, M.S. *Exhibited February* 28, 1902.

A MAN æt. 38 shows the recent scar of an incision reaching from the right loin below the twelfth rib at the outer border of the erector spinæ, over the crest of the ilium, to about the middle and just above Poupart's ligament.

At the operation, on January 9, there was an abscess extending from the liver downwards, behind and external to the ascending colon and cæcum, into the pelvis, containing foul pus and gas.

Within three years an abscess had formed three times before, and had been incised or had been allowed to burst through the scar.

The incision gave room for the evacuation of the abscess, the breaking down of its loculi, and also for the removal of the diseased appendix, which was buried in inflammatory tissue

behind the cæcum and ascending colon. The cavity was then filled with iodoform gauze. A week later healthy granulations lined the cavity, and the temperature had become normal. The whole wound was therefore sutured, and second union has followed.

XXXIV.—*Case of Paralysis of the Cervical Sympathetic.* By Purves Stewart, M.D. (introduced by W. G. Spencer, M.S.). *Exhibited February* 28, 1902.

THE patient, J. A. P., a soldier, was wounded fourteen months ago by a Mauser bullet, which entered the left side of the neck one and a half inches below the mastoid process, and came out in the seventh right intercostal space in the posterior axillary line, lodging in his bandolier. The bullet thus passed in front of the vertebral column, injured the brachial plexus on the right side, and wounded the right lung. He had temporary total paralysis of the right arm, which gradually cleared up. There was some hæmoptysis and slight dysphagia for a few days only. Ever since the injury the sight in the right eye has been less acute than in the left, and he has noticed that he does not sweat on the right side of the face, scalp, and neck, or on the right upper limb.

Present condition.—There is slight weakness of the small muscles of the right hand, and an area of slight analgesia along the inner border of the upper arm, forearm, and hand, including one and a half fingers, with a small patch in the axilla. The lower limbs and the left upper extremity are normal.

The right pupil is smaller than the left, and does not dilate when shaded. Both pupils react briskly to light and on convergence. The cilio-spinal reflex is absent on the right side. The right palpebral fissure is narrower than the left, and the right eye is slightly sunken in. He does not sweat on the right side of the face, scalp, neck, and upper part of thorax, nor on the right upper extremity. This area of anidrosis is bounded by the middle line, and extends down as far as the third rib in front, and posteriorly as far as the middle of the scapula.

The lesion is apparently one of the first and second dorsal nerve-roots on the right side.

DESCRIPTION OF PLATE XXII.

To illustrate Dr. Purves Stewart's case of Paralysis of the Cervical Sympathetic.

Powdered charcoal was puffed upon the patient's skin whilst sweating. On the right side observe the absence of sweating—the anhidrotic area being bounded by the middle line; the narrowing of the palpebral fissure; the smallness of the pupil.

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FIG. 2.

FIG. 1.

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DESCRIPTION OF PLATE XXIII.

To illustrate Dr. Purves Stewart's case of Paralysis of the Cervical Sympathetic.

FIG. 1 shows the area of anhidrosis in lesions of the cervical sympathetic. This includes the scalp, face, neck, upper part of thorax, and upper limb. The black line on the upper limb marks the limits of an analgesic area.

FIG. 2.—Showing the area of anhidrosis in a lesion of the cervical sympathetic. This includes the head and neck, the upper part of the thorax, and the entire upper limb. The black line on the upper limb marks the analgesic area from concomitant lesion of the brachial plexus.

XXXV.—*Splenic Anæmia with a Splenic Bruit.* By H. D. Rolleston, M.D., and Lawrence Jones, M.B. *Exhibited February* 28, 1902.

A MAN æt. 22 years was under treatment twelve years ago with splenic enlargement, but has had more definite symptoms for the last four years. There is no history pointing to syphilis or malaria. Three years ago he had an attack of epistaxis, and since then he has had repeated attacks of epistaxis, hæmatemesis, and melæna.

He has considerable enlargement of his spleen. The blood shows anæmia of what is usually called the chlorotic type. Red blood-corpuscles 2,400,000, Hg. = 25 per cent., and some diminution of the leucocytes; the highest number counted after a febrile attack (? influenza) being 4600 per c.m., the lowest 3500. There is apparently no change in the relative numbers of the varieties of leucocytes.

There is a distinct blowing continuous murmur, resembling the bruit de diable, to be heard over the spleen. This is most distinct at the back in the ninth space, just outside the scapular line; and in front in the ninth space, just posterior to the anterior axillary line. Its intensity is greatest when the patient holds his breath in full inspiration. There is no thrill. It is suggested that the murmur is venous, and is produced either in the vasa brevia from torsion of the gastro-splenic omentum, or in some of the branches of the splenic vein in the hilum of the organ as a result of a temporary kink. According to Harris and Herzog ('Annals of Surgery,' July, 1901, p. 134), there is enormous dilatation with thinning of the walls of the splenic vein in splenic anæmia. There are conditions which are favourable to kinking, and so to the production of venous murmurs.

A case of enlarged spleen due to malaria, with a bruit and thrill, was shown before this Society in 1899 by Dr. R. Crawfurd (vide *Transactions*, vol. xxxii, p. 234). In a case of splenic anæmia recorded by Sippy (*American Journ. of Med. Sciences*, vol. cxviii, p. 430, 1899) a bruit was audible over the enlarged spleen.

Postscript.—Splenectomy was performed by Mr. Shield on March 24, but death from gastro-intestinal hæmorrhage followed on the second day; no autopsy could be obtained.

XXXVI.—*Myopathy (Atrophic and Pseudo-hypertrophic) associated with Peculiar Pigmentation of the Legs.* By S. Vere Pearson, M.B., and H. D. Rolleston, M.D. *Exhibited February* 28, 1902.

History.—A clerk æt. 22. Eight years ago had scarlet fever, in the second week of which there were rheumatic pains and swellings in the knees. Weakness in the back noticed a week later, followed by wasting of the thighs. Steady progress of the disease since onset. No similar affection in his relations. Pigmentation of the legs noticed for one year. Has never taken medicine continuously for more than six weeks, and believes he has never taken arsenic. He cannot explain the pigmentation, though at one time he stood in front of the fire for rather long spaces of time. No similar affection in any of his brothers and sisters, or in their children.

Condition.—Muscles: atrophy of both thighs, tibiales antici, ext. commun. digit. ped., peronei. Slight foot-drop, talipes equinus, and pes cavus. The sternal halves of both pectorals, the latissimi dorsi, teres major, subscapulares, and erectores spinæ are wasted, and there is lordosis.

The gastrocnemii, infraspinati, and deltoids are large. Although most of the muscles show A.C.C. > K.C.C. there are no other reactions of degeneration.

Sensation is normal.

Pigmentation: the skin of the legs is uniformly discoloured, and is a reddish-brown. Beginning above indefinitely, it becomes more intense, until it abruptly stops just above the ankle, where the tops of the boots would come. Below this level there are some scattered discrete patches of yellowish-brown pigmentation, with a few patches of rather a redder colour, on the dorsum of the feet. As to the causation, it is improbable that the pigmentation is due to exposure to the heat of a fire, inasmuch as there is pigmentation on the feet. There is no reason to think he has Addison's disease; the distribution does not agree with that of the muscular atrophy. The question arises as to whether there is a degenerative change in the skin leading to pigmentation.

DESCRIPTION OF PLATE XXIV.

To illustrate Dr. Ormerod's case of Eruption of Psoriasis Type.

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DESCRIPTION OF PLATE XXIV.

To illustrate Dr. Ormerod's case of Eruption of Psoriasis Type.

XXXVII.—*Eruption of Psoriasis Type, probably Syphilitic.* By J. A. Ormerod, M.D. *Exhibited February* 28, 1902.

Annie C., æt. 39, married, came to St. Bartholomew's January 28, 1902, complaining of a rash, not differing materially from that which is now seen, and ulceration of the leg now healing.

Rash affects face, head, trunk, upper arms, and thighs mainly, other parts in a less degree. Mostly consists of raised red papules and patches, the majority of which are covered by scales. On the face there are no scales, and when first seen the spots on the face, and indeed the whole face, had an œdematous appearance, and at one point there was slight ulceration, since healed. On the back there is a certain symmetry of arrangement, the papules running down from the spine around the waist. On each forearm there is a pair of spots very symmetrically placed. The points of the knees and elbows are quite free. Some spots on the scalp are discrete, and covered with thick adherent scales as in ordinary psoriasis. On the vertex there is a more diffuse scaliness. On the upper third of the left leg were three ulcers; the largest, the size of a florin, was inflamed and unhealthy-looking when first seen. The fauces, when first seen, were somewhat red and swollen, and the uvula œdematous, but there was no ulceration.

She says that the rash came out on the second day of an illness characterised by shiverings and cold feelings, aching in the bones and in the head, and sore throat. This was at the end of last October, and she was in bed three weeks. The rash, she says, has not altered much since, except that it got worse soon after getting up, when she "caught a cold." The rash has itched.

Family and previous history unimportant; one child which died young, twenty years ago. No miscarriages.

Treated with Liq. Hydrarg. Perchl. internally, and Lotio Nigra to the leg. The ulcers on the leg are nearly healed, the throat is no longer sore, and the face has improved a little; but in the rash as a whole there is no difference.

After being shown to the Society, this patient was put on mercurial inunctions, and iodide of potassium by the mouth, and she got rapidly well.

XXXVIII.—*Atrophic Paralysis of Hands and Wrists, being the remains of a recurrent generalised paralysis (? Peripheral Neuritis).* By J. A. Ormerod, M.D. *Exhibited February* 28, 1902.

WALTER C., æt. 28, formerly in Dragoon Guards, invalided for present complaint.

Admitted to St. Bartholomew's October 16, 1901; he then had *marked weakness and wasting of the upper limbs* from the scapulæ downwards, viz. of the following muscles:

Deltoids, spinati, biceps, triceps (less); extensors of wrists and fingers, flexors (less); intrinsic muscles of hands. Pectorals, trapezii, serrati, unaffected.

In the lower limbs, *some wasting of thighs*, legs normal, knee-jerks feeble, plantar reflexes normal. No affection of sphincters, no affection of sensation.

Electrical reactions normal, but feeble in some of the affected muscles; R. D. in right deltoid and the interossei of both hands.

Has now much improved, and the paralysis is limited to the extensors of wrists and fingers, and the intrinsic muscles of the hands; but even these are better.

This is the fourth attack of the kind.

First attack in March, 1899, while serving at Sialkote in the Punjaub; abdominal pain and constipation; pain back and arms, and vomiting; pain legs. Wasting followed. Well in two months, except for some loss of power in fingers, and (?) adductors of thighs.

Second attack July, 1899, less severe. Well by October, except as to fingers. Home to Netley; discharged March, 1900, on account of fingers.

Third attack May, 1901, cough and cold, abdominal pain and constipation, some fever (101°). Weakness and wasting noticed after a week, but not wholly incapacitated.

Fourth (present) attack began like third, but the paralysis has been so bad that he could neither feed nor dress himself.

Other illnesses.—Chancre in 1893; slight malaria, but not enough to lie up, when in Bengal (this was prior to present illness); dysentery on voyage home (after present illness).

There was no similar case in his regiment.

It may be added that this patient's urine darkens, after

standing, to an intense brownish black. This appears to be due to some rare kind of pigment. It is under investigation by Dr. Archibald Garrod.

XXXIX.—*A familial type of Paralysis allied to the Myopathies and to Friedreich's Disease.* By F. E. Batten, M.D. *Exhibited February* 28, 1902.

THE father and mother of the family are Polish Jews, and are third cousins, *i. e.* grandchildren of two brothers. They have five living female children. The mother has had two male children born after the fourth living child. One died shortly after birth and the next at five months. There is some evidence pointing to syphilitic infection at this time. The fifth child, a girl, exhibits no sign of specific disease.

G. K., a girl æt. 15, is the eldest member of the family. She has always walked slowly, and has been weak on her legs, and has frequently fallen down, but it is only during the past *nine months* that she has become obviously weaker on her legs. She works well with her hands, but is a slow writer. The weakness in the legs is most marked in the peroneal and anterior tibial muscles, and in the extensors of the toes. There is foot-drop and pes cavus. There is practically no wasting. The back is straight, and there is no weakness of the upper extremities or face. There is considerable myopia; both pupils react well to light and on accommodation; the fundus is normal. There is slight nystagmus on lateral deviation. The knee-jerks are absent, and the plantars cannot be obtained.

No reaction to a strong faradic current can be obtained in the muscles of either leg, not only in the muscles obviously affected, but also in those not obviously weak. The arm muscles just react to a strong faradic current, and even the muscles of the face react badly. To galvanism there is no reaction to either the anodal or kathodal current in the anterior tibial, peroneal, or extensor muscles of the toes, but a slight contraction is obtained in the gastrocnemius.

E. K., girl æt. 13, brought at my request, was said to be all right. Runs and walks well; marked pes cavus, great toe drawn back and flexed at terminal phalanx. Unable to dorsi-

flex the foot beyond the right angle, and unable to extend the small toes.

Knee-jerks are obtained on reinforcement. Plantars not obtained.

Electrical reaction.—No contraction can be obtained in the long extensor of the toes or peronei muscles to a strong faradic current. A fair contraction can be obtained in the anterior tibials and quadriceps extensor, but the gastrocnemius gives a poor contraction.

T. K., girl æt. 11, brought for weakness of her legs, first noticed *eighteen months ago.* Walks and runs fairly well. Feet in position of pes cavus. Unable to dorsiflex the feet beyond the right angle. Knee-jerks present, not active; plantars give flexor response. There is slight tremor of the hands, but no ataxia; there is no nystagmus; there is myopia. She is in the fifth standard at school.

Electrical reaction.—Almost no reaction in the anterior tibial, peroneal, or gastrocnemius muscles of the legs. No reaction obtained in the extensor muscles of the toes.

R. K., girl æt. 9, brought to hospital at my request. Runs and walks well, gets up from floor well. She is in the second standard, and is the only member of the family who is backward. Myopic.

Knee-jerks active; no pes cavus; can dorsiflex the foot, but cannot extend the small toes well. A reaction is obtained to a strong faradic current in the anterior tibial, peronei, gastrocnemius, and extensor brevis digitorum, and the extensor longus digitorum on the left side, but no reaction can be obtained in the extensor longus digitorum of the right side.

D. K., girl æt. 4, shows nothing abnormal except that she is unable to dorsiflex the small toes. All muscles of the leg except the extensor longus digitorum react to a fairly strong faradic current.

XL.—*A Case of Probable Hydromyelia in a Child.* By F. G. Penrose, M.D.. *Exhibited February* 28, 1902.

CHARLES T., æt. 6 years.

A first child born at eight months; labour easy, vertex presentation. Three children born since, all healthy; no miscarriages. Both parents healthy.

Nothing wrong was detected with the infant until he was eleven months old, when it was noticed that he did not use his arms, and that his eyes were "wobbly."

He has never stood up.

On January 4, 1902, was admitted into St. Thomas's Hospital in a very collapsed condition suffering from pneumonia. On January 21 he was transferred to the South-Western Fever Hospital, under suspicion that he might have recently had scarlet fever, as he was found to be peeling, and a sister had returned from the Northern Fever Hospital in December, 1901, after scarlet fever.

I am indebted to my friend Mr. A. S. Morley for the opportunity of seeing the child, and also for the notes of the case during the child's stay in the South-Western Fever Hospital, from which the following abstract is largely drawn.

The child's head is small. Measurements: circumference, eighteen and a half inches; across from one external auditory meatus to the other, twelve inches; from glabella to external occipital protuberance, thirteen and a quarter inches.

Intelligence is apparently below normal. He is rather shy, and only rarely attempts to speak. He is readily made to laugh, and hardly ever cries; he is distinctly of a happy disposition. He is completely helpless to change his position; and if set up, his head falls forwards, so as almost to allow his chin to touch his chest; if unsupported he falls forward in a heap. He has never had any real incontinence of urine or fæces, but frequently has "accidents," owing to his not stating his wants clearly.

The organs of special sense appear to be normal, and there is no paralysis of any cranial nerve to be detected.

Nystagmus, mostly though not entirely lateral, is present, irregular and intermittent, with very short, rapid oscillations.

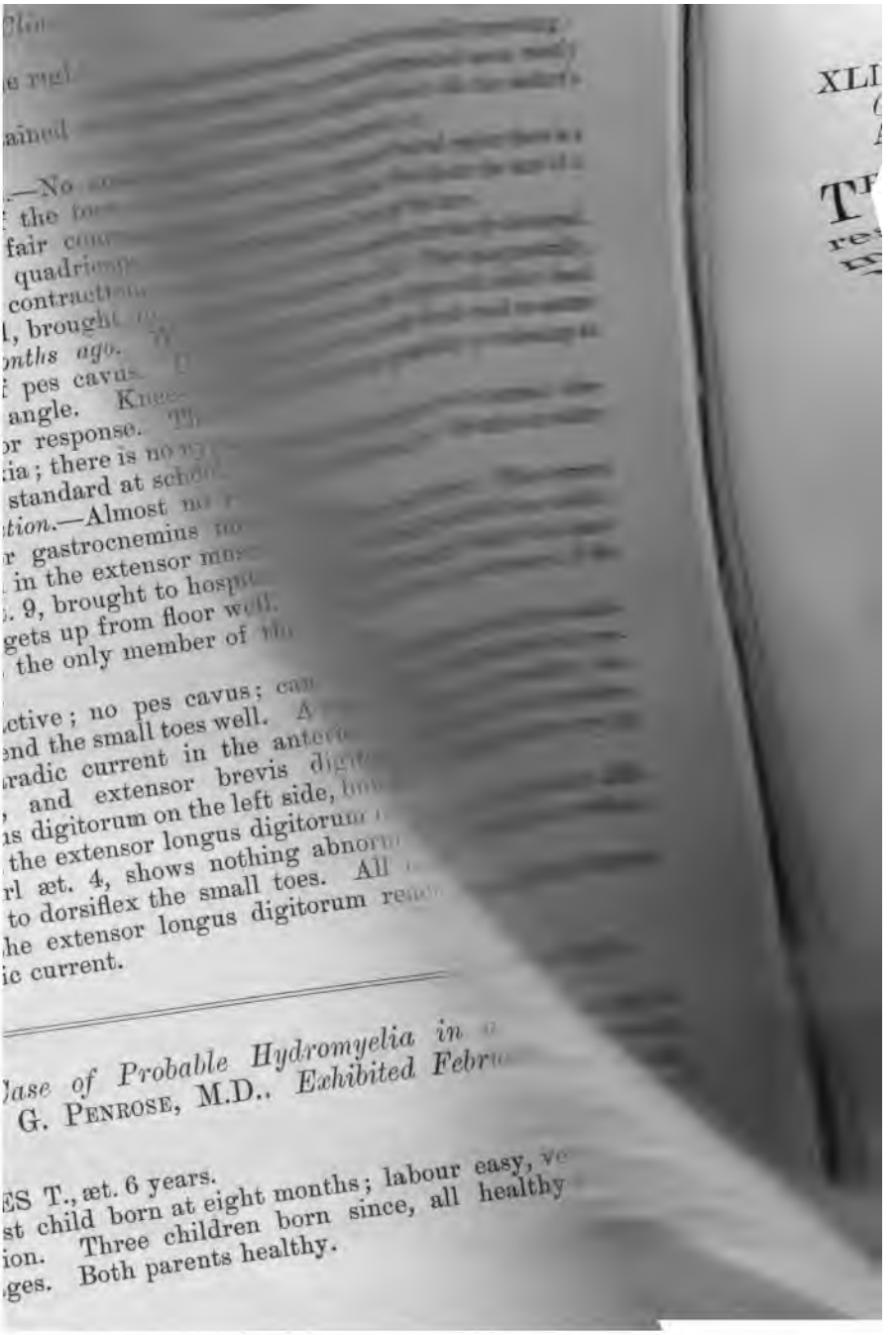
The limbs have always been weak, and the child has never attempted to walk.

There has been great loss of sensation—at any rate to pain—in all four limbs.

He has had the habit of biting his fingers till they bled and drinking the blood. (It is in this way that the mother accounts for the peculiar clubbing of the finger tips.)

He has also frequently burned himself by leaning against hot fire-guards, pipes, etc., and has never felt any pain.

He is very liable to get sores from sitting or lying in one position.



(The mother thinks that sensation is steadily improving.)

There are numerous large non-pigmented scars, mostly situated on the extremities, which bear out the mother's statement of his liability to get burnt, etc.

In the lower lumbar and upper sacral region there is a round pigmented scar in the middle line about the size of a shilling, partially surrounded by long fine hairs.

The extensor muscles of the spine are poorly developed. The arms are thin and easily get cold. They hang naturally, and the child can pick up a piece of sugar with either hand and transfer it to the other; but both hands tend to assume the position of ulnar palsy when grasping or releasing an object.

Sensation.—Of face and neck appear to be normal; elsewhere, when tickled he laughs readily. He takes no notice of a vigorous pin-prick.

Electrical reactions.—Interrupted current. (The current used was strong enough to be decidedly painful to an adult.) All muscles respond, most readily enough, though the quadriceps extensors of the thighs and the long extensors of the back less readily than others.

Whilst the lower limbs were being tested (and the examination was commenced with them) the child watched the proceedings, and gave no sign of feeling any discomfort, but after passing up the trunk, as soon as the level of the shoulders was reached, he immediately began to wriggle and to cry out a little.

Reflexes.—Though not absent, are only elicited with difficulty on the trunk and extremities. Eye and face reflexes normal.

Remarks.—The diagnosis has to be made, I think, between the following conditions:

(*a*) Hydromyelia or syringomyelia.

(*b*) The infantile type of idiopathic muscular atrophy.

(*c*) Mental deficiency from microcephaly.

The remarkable deficiency of appreciation of heat and of pain, whilst retaining ordinary tactile sensation, appear to point very strongly to the case belonging to the first group, and the scar in the lumbo-sacral region is suggestive of developmental anomaly of the spinal cord.

XLI.—*Case of General Paralysis of the Insane in a Girl aged nine.* By C. H. FENNELL (introduced by A. GARROD, M.D.). *Exhibited February* 28, 1902.

THE father had syphilis before marriage, and died last year in Hanwell of paralytic dementia. The patient is the result of the third pregnancy, the first two having produced miscarriages: two healthy children were subsequently born. No definite history of congenital syphilis could be obtained; the child was mentally bright up to the onset of symptoms six months ago, when she gradually became apathetic and dull. Her speech is slurring. There is a congenital squint; the pupils do not react to light, and contract but feebly with accommodation. Bilateral optic atrophy is present with numerous patches of old retino-choroiditis. The facial muscles show intention tremor, and the tongue is tremulous on protrusion; the gait is hesitating and slightly ataxic. The knee-jerks are exaggerated; there is no sensory defect.

XLII.—*Two Cases of Filariasis, one treated by a new operation, the other untreated.* By PATRICK MANSON, M.D., and RICKMAN J. GODLEE, M.S. *Exhibited February* 28, 1902.

CASE 1.—Man æt. 31. Born in Calcutta. Civil engineer. Lived in India till 1898, then went to Gold Coast. Filariæ discovered at the end of 1900, but has had slight swelling in groins for five or six years. The testicle (? scrotum) has also swelled.

Patient was sent to me by Dr. P. Manson, November, 1901. He had a large swelling (varicose lymphatics) in right groin. There were a few filariæ in the blood.

On December 4, at Dr. Manson's suggestion, I cut down on the lower part of the tumour, and with much difficulty anastomosed a dilated lymphatic vessel with a tributary of the internal saphenous vein. The fluid in the glands and lymphatics was a mixture of lymph and chyle. The result

has been the almost complete disappearance of the lower part of the swelling.

The upper part of the swelling remained. In January, 1902, I anastomosed one of the dilated lymphatic vessels of the cord with one of the spermatic veins. The operation was easily performed. The lymphatics contained pure lymph. The swelling has diminished, and apparently is continuing to diminish.

It is probable that the obstruction to the thoracic duct is opposite the lymphatics accompanying the spermatic vessels, and thus, although there is an obstruction to the return of lymph from their radicles, there is no regurgitation of chyle.

Case 2.—A man æt. 20, from Hayti, who had much larger swellings in both groins than the first patient had exhibited before he was operated upon, some of the varicose lymphatics extending as low as the middle of the right thigh.

Since this patient was shown he has been submitted to two operations. At the first the lower part of the swelling in the right groin was exposed, in part behind, and a lymphatic was anastomosed with a vein. It was a more difficult and less satisfactory operation than that performed in Case 1. The lymphatic spaces were larger; the fluid was of an opaque pink colour. At the second operation, which was still more difficult, a large part of the swelling on the left side was cut away, and a lymphatic was joined to a tributary of the internal saphenous vein close to its termination. The varicose lymphatic in the middle of the right thigh was removed at the same time. The operation was followed by great shock; there was much oozing of mixed chyle and lymph. As in all the operations on both patients, however, the wound healed rapidly. There does not at present appear to be so much improvement in this case as in the other one.

XLIII.—*Left Parietal Meningocele or Cephalhydrocele.* By H. L. Barnard, M.S. *Exhibited April* 25, 1902.

THOMAS C., æt. 2 weeks, was brought to the London Hospital for a swelling on his head. The swelling was noticed two days after birth, and is not increasing in size.

Bale and Danielsson, Ltd.

DESCRIPTION OF PLATE XXV.

To illustrate Mr. Barnard's case of Multiple Chondromata or Osteomata, with Synostosis of the Lower Epiphyses of both Ulnæ.

The delivery was not instrumental or difficult. The swelling is about the size and shape of half a small egg. It is situated on the upper and posterior part of the left parietal bone, and is distinct from the anterior and posterior fontanelles and the sagittal suture. A bony edge can be felt around its site on the skull. It fluctuates, and becomes more tense on crying and coughing. An attempt to reduce it led to cessation of respiration and pallor. It has not been punctured.

Note.—The tumour entirely disappeared in a month, and was therefore probably a cephalhæmatoma.

XLIV.—*Multiple Chondromata or Osteomata, with Synostosis of the Lower Epiphyses of both Ulnæ and Fibulæ.* By H. L. BARNARD, M.S. *Exhibited April* 25, 1902.

ALBERT M., æt. 3 years, shows the following condition:—Tumours are found projecting from the ribs, scapulæ, clavicles, and femora, etc.; they are bony, hard, knob-like, and firmly fixed to the bones. The epiphyses are for the most part enlarged. The lower epiphysis of the ulna has undergone early synostosis on both sides, leading to ulnar displacement of the hand at the wrist-joint. The fibulæ (the homologous bones in the leg) are in the same condition. The internal malleolus is on the same level or a little lower than the external, and the foot is displaced outwards, as in talipes valgus. For this condition he has to wear irons. He has suffered from severe rickets.

XLV.—*Tumour of Abdominal Wall.* By H. L. BARNARD, M.S. *Exhibited April* 25, 1902.

ALICE B., æt. 45 years, has had a large tumour of her abdominal wall for many years. It has undergone no change for a great length of time. It is the size of a large orange, lobulated, very hard, uniform in consistency, and

situated to the right of the umbilicus. When the patient raises her shoulders from the couch the tumour is partly fixed, and becomes more prominent. She has also an umbilical hernia. The tumour is probably a fibroma or fibro-lipoma.

XLVI.—*A Case showing Conspicuous Delay of Growth and Development.* By HASTINGS GILFORD. *Exhibited April* 25, 1902.

THIS man is an instance of a disorder which has not hitherto been systematically described, though some dozen or more cases have been recorded by French and German writers. He is twenty-eight years of age, and comes of a healthy and well-grown family. It was first noticed that he was not growing when he was between one and two years old. I first saw him just five years ago. He was then 1·078 m. (three feet six inches) in height, and has since grown 18 mm. His height and proportions resemble those of a boy of between six and seven. A radiogram of the hand shows that the ossification is no farther advanced than is usual at the age of ten, while the dentition is like that of a child of the same age, though the teeth are crowded and irregular. The contour of the body and the appearance of the head and face are all childish. But the sexual organs are the most backward in development, for the testes have not descended, and the external genitals are infantile. There is no hair on the body or face. The intelligence is good, but his behaviour and conversation are more like those of a child than of an adult. The voice is more treble than that of an adult, though it is not quite childish in tone and pitch. The thyroid gland can be felt.

The dwarfism in this case is apparently not due to any of the known causes of that condition. Achondroplasia, rickets, and microcephaly can be definitely excluded.

There seems to be no indication that the imperfect development is due to heart disease, syphilis, or defective action of the thyroid gland, or hypoplasia of the sexual organs. The condition is certainly not one of normal infantilism.

XLVII.—*A Case of Ethmoidal Mucocele.* By ANTHONY A. BOWLBY. *Exhibited April* 25, 1902.

JOHN A. W., æt. 26, horsekeeper. When five years old he suffered from "an abscess in the left eye," presumably from the history referring to a swelling in the orbit above the inner canthus and closely adherent to the inner wall. This condition lasted for thirteen months, and was followed by a persistent swelling in the region of the upper part of the nose on the left side, remaining constant in size and free from pain.

The swelling in the inner side of the right orbit he remembers to have had in its present state for the last ten to fifteen years; he has suffered no inconvenience from it whatever.

On March 10, 1902, he first noticed that he was unable to breathe through his left nostril, and at the same time he experienced pain at the inner side of his left eye, and noticed the swelling there to be increasing in size. Since then this swelling had progressively increased in size, become more tender, and accompanied by greater pain.

He has never had headache, vomiting, or diplopia, and sees as well with one eye as the other.

XLVIII.—*Case of Congenital Absence of Nose and Anterior Nares, with highly arched Hard and Soft Palate.* By Dr. ESSEX WYNTER. *Exhibited April* 25, 1902.

A FEMALE infant, six weeks old, the first child of healthy parents, showed this very unusual deformity without "harelip" or deficiency of præmaxilla. The child suffered from difficulty in feeding and respiration. There was no other deformity, but the child was ill nourished. When subsequently admitted to the Hospital for Sick Children, Mr. Bernard Pitts ascertained that a bent probe could only be passed forward half an inch behind the soft palate. An

attempt to open the nostril resulted only in forming a communication with the mouth behind the alveolus, the site of the nasal cavity being apparently occupied by solid bone.

XLIX.—*Probable Atelectasis from Birth.* By Sir Hugh Beevor, Bart., M.D. *Exhibited April* 25, 1902.

RIGHT lung, upper lobe appears to be represented on the surface of the chest only by a small area against the clavicle. Absence here of evidence of fibroid disease and of pleural thickening suggests collapse, acquired or congenital. Double coloboma of iris and sector-shape opacities of lens, pointed out by Mr. Haydon, perhaps also the high and narrow palate, are of congenital origin. These deficiencies seem to render it probable that the case is one of atelectasis from birth. Heart, no signs suggesting intrinsic disease.

B. M., æt. 24, housework. Under observation three weeks in Victoria Park Hospital. Patient is small and of apparently feeble physique; yet her family history reveals no weakness, and her previous history is one of exceptional health, diarrhœa being the only ailment her mother can name in childhood.

B. M. sought advice for pain in the chest; this was alleviated by treatment for dyspepsia.

The left chest but slightly exceeds the right in size and extent of expansion. The heart is found to be displaced, the apex or maximum impulse being felt in the right axilla, in the third interspace, and five and a half inches from mid-sternal line. In the first three interspaces pulsation may be seen and felt; it is also just palpable under left breast. Dulness extends from sternum to mid-axilla, and from third interspace downward; above this interspace is a less-degree dulness, especially less over the inner portion of the first two spaces and above the clavicle. Percussion elsewhere shows a slight impairment behind from the middle of scapula upward and to axilla. Cardiac auscultation reveals a faint systolic murmur, heard at the base, and considered due to the displacement only. Pulmonary auscultation shows a very faint respiratory murmur above the abnormal cardiac dulness,

without evidence of a consolidation with patent bronchi. Vocal fremitus equal above clavicles, absent below right. No clubbing of fingers. Slight scoliosis, slight beading of ribs. Other organs found normal.

Dr. Martin has taken a skiagram. He observed by the screen that the heart was upon the diaphragm.

L.—*Case in which Excision of the Right Wrist by a Posterior Median Incision for Tuberculous Disease was done three years ago.* By W. G. SPENCER, M.S. *Exhibited April* 15, 1902.

THE patient, then æt. 23, was seen with Dr. A. Cope, suffering from tuberculous disease of the right wrist, which had already broken down, the wrist-joint, the carpus, and the tendon sheaths of the lower forearm being involved. Excision of the wrist-joint and carpus by a posterior median incision was done in April, 1899. In March, 1900, some sinuses in the right forearm were scraped, and arthrectomy of the left knee performed. This was followed by definite healing.

The patient, who is an accountant, has regained his previous good handwriting, and can lift and carry light objects.

LI.—*A Case of Congenital Morbus Cordis, with Extreme Clubbing of the Fingers and Toes.* By J. WALTER CARR, M.D. *Exhibited April* 25, 1902.

THE patient is a girl æt. 10, who is said to have been always more or less "blue." She has never been able to take any active exercise on account of shortness of breath, and feels the cold so much that she can only attend school in the summer. She suffers occasionally from giddiness and præcordial pain, but has never had any œdema.

She shows very marked cyanosis, particularly of the fingers, toes, cheeks, lips, and tongue. There is extreme clubbing of the finger ends, and also of the toes, especially the great toes, but a skiagram of the fingers shows that the distal ends of the third phalanges are not enlarged. In spite of the high degree of cyanosis, it is notable that the child's development and general nutrition are fairly good.

The nature of the cardiac lesion is obscure. The heart is considerably enlarged transversely, especially to the right; the apex-beat is best felt in the fourth space, just outside the nipple line (but probably corresponds with the fifth rib). There is well-marked pulsation over the lower end of the sternum, and dulness extends from over an inch outside the right border of the sternum to just outside the left nipple line. A rather faint systolic murmur is heard over the base of the heart and at the inner ends of the adjacent intercostal spaces on each side. The heart intermits irregularly at times. The pulse is of distinctly high tension for a child.

The blood shows a considerable degree of concentration, containing 8,000,000 red corpuscles per c.mm., and 120 per cent. of hæmoglobin.

All the other organs and the urine are healthy. The temperature is not subnormal.

LII.—*Arthropathy in a Case of Bronchiectasis.* By Percy Kidd, M.D. *Exhibited April* 25, 1902.

GEORGE V., æt. 30, compositor, the subject of bronchiectasis, with profuse foul expectoration, presents the following condition of joints:—Knees, wrists, and ankles enlarged and contain fluid. Hands and fingers very large, the latter extremely clubbed. The patient says that his hands have always been large, but he is unable to state whether they have increased in size or not. The toes also large and clubbed. Skiagrams show no definite bony change.

History.—He states that he has had "gout" on several occasions during the last six years, but the present joint

affection began three months ago with pain and stiffness in the knees and ankles. He has had a chronic cough and expectoration for three years, and nearly a year ago he noticed that the sputum had a foul taste, but he was not aware of any offensive smell from it.

Soon after his admission to the hospital about a fortnight ago the expectoration became profuse and fœtid, and the cough assumed a paroxysmal character. No tubercle bacilli can be discovered in the sputum.

LIII.—*A Case of Urticaria Pigmentosa.* By Arthur Bevan (introduced by Archibald E. Garrod, M.D.). *Exhibited April* 25, 1902.

THE patient is a girl æt. 4, under the care of Dr. Garrod. The eruption first appeared on the legs at the age of two or three months, the spots "looked like flea-bites," but did not fade, and gradually became brown in colour. At six months old a fresh crop of spots appeared suddenly on the abdomen, and the eruption has since spread gradually over the whole body, reaching within the last few months to the margins of the face. There has been no irritation of the skin, and the general health has always been good.

At the present time the skin of the entire body, including the neck, forehead, and marginal parts of the cheeks, is covered with closely set papules varying in size from a pea to a threepenny piece. The colour for the most part is a dull pink, or in places brownish, while about the face the spots are faint and have a lemon-yellow tinge.

The eruption does not fade on pressure, and is not raised, but on friction of the skin, especially of the back, the spots become distinctly elevated. There are no wheals. No signs of irritation are present. There is no pigmentation of the mucous membranes. The blood-count shows nothing abnormal, and there is no sign of visceral disease.

LIV.—*Chancriform Ulcer of the Tonsil, or "Vincent's Angina."* By StClair Thomson, M.D. *Exhibited April* 25, 1902.

A BOY æt. 12 was shown, illustrating an uncommon form of membranous ulceration of the tonsil which has received little attention, although it has been described on the Continent under various names, such as "diphtheroid angina," "membranous amygdalitis," "ulcerating lacunar tonsillitis," and the "angina of Vincent." The latter name was given to it in consequence of the discovery by Vincent in the membrane of a spindle-shaped bacillus of 4 μ in length and 1 μ in width. He had previously discovered the same bacillus in the dirt of hospitals (Vincent, *Annales de l'Institut Pasteur*, 1895, p. 488).

The boy had only been complaining of a little sore throat for four days. The right tonsil was seen to be occupied by an oblong, greyish slough; the margin was vertical and not inflamed. The slough was made up of a pulpy or chalky membrane, which was easily detached, and exposed an irregular, ulcerating surface, which bled readily. The rest of the throat presented nothing abnormal. The glands below the angle of the right jaw were somewhat enlarged and painful. The margin of the ulcer was not hard to the touch. The temperature was only 99°, and the pulse 84. There was, beyond a slight soreness of the throat, no general discomfort.

Time had not allowed of a bacteriological examination.

The similarity of the condition to a syphilitic chancre was obvious.

P.S.—The bacteriological examination did not reveal Vincent's bacillus. Under expectant treatment the condition improved so rapidly that at the end of a week the patient withdrew from further observation.

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